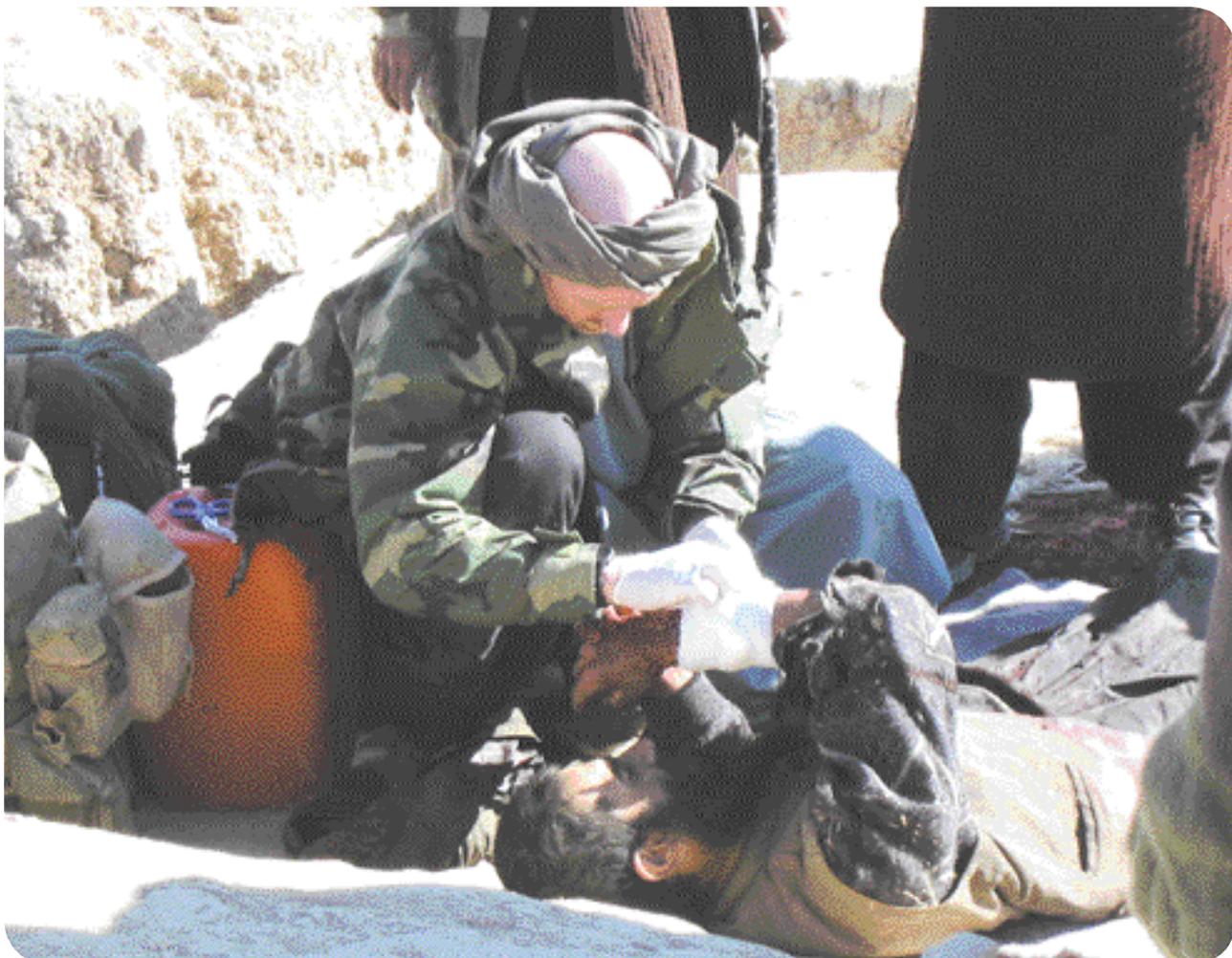


Volume 2, Edition 3

Summer 2002

Journal of Special Operations Medicine

A Peer Reviewed Journal for SOF Medical Professionals



Dedicated to the Indomitable Spirit & Sacrifices of the SOF Medic

From the Surgeon



Greetings once again from Tampa and the HQ/USSOCOM. We here working to support the medical folks caring for our Soldiers, Sailors, Airmen and Marines continue to be proud and awed by the professionalism demonstrated on a daily basis by our folks around the world. Your excellence pushes our commitment toward improved training and medical equipment for our Medics, Corpsmen and PJs.

Most recently, the Biomedical Initiatives Steering group met and we are moving forward on several fronts;

1. Hemostatic Dressings: For some time now we have supported the fielding of this technology and we may in fact have one in the field by this Fall (two types are being pursued-the Fibrin Dressing and the Chitosan Dressing).
2. Technology Panel: A Tactical Combat Casualty Care Panel is being assembled including trauma experts and medical operators from all the Services to look at trauma resuscitation and help with algorithms to fluid use (crystalloid versus colloid versus blood versus new technology), vascular access (intravenous versus interosseous routes), better airway tools and management, and other issues. They will also be looking at pushing the technology toward the fielding of an "artificial blood" or HBOC.
3. Lessons Learned: The BISC is funding a gathering of our medical folks who were deployed and have experiences in field medicine under combat conditions. We feel it is critical to hear the stories and adjust our training and equipment as the scenarios dictate. The experiences will be chronicled and the data will be shared with all our SOF medics worldwide.

In addition to these issues, we continue to look to make SOF military diving operations safer and better, improve disease detection and surveillance, and learn from the messages we get from the experiences of our operators.

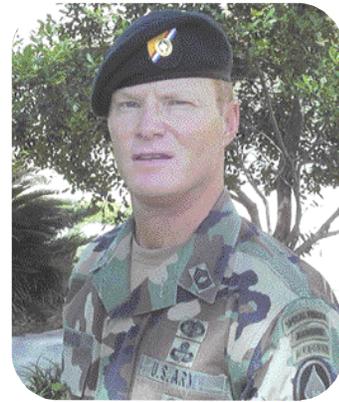
Finally, we need to "foot-stomp" the medical surveillance issues. Very soon we hope to have a hand-held computer in the hands of all our medical folks which will assist them in Pre-deployment data, Post-deployment data, point of service medical encounter data (600's) and at the same time provide a reference tool with the SOF Medical Manual, treatment algorithms, PDR data, etc. USSOCOM is way out in front of the Services on this Congressional mandate toward surveillance. When the tool arrives, use it, gather the data on your folks and then deliver it to us (the component Surgeons and us at SOCOM) and we will assure that it gets to the medical records and the data will be assembled to benefit SOF deployments in the future and perfectly monitor the exposures and encounters of the war-fighters we are sworn to protect.

Again, you make us all very proud, "keep on keeping on" and let us know how we can make it better for you.

dhammer

Dave Hammer (center) ,1st Marines, Fleet Marine Force Pacific, winter 1959.





SENIOR ENLISTED MEDICAL ADVISER (SEMA), MSG
Michael A. Brochu

From the ROAD DOG in the BIG HOUSE,

This quarter has flown by so let me update you on where we stand at the publishing of this journal. The USSOCOM Command Surgeon's office is more active than ever fighting for you the SOF Enlisted Health Care Provider. Here is a brief discussion of the top topics:

Health Surveillance- USSOCOM has a practical (40%) health surveillance solution, which meets operational and statutory requirements. Phase two procurement will give the SOF medic the ability to document encounters from the point of injury back through the echelons of care. The device will assist the medic by providing an up to date treatment modality, re-order the expended class VIII supplies, complete the FMC / SF-600, and provide the needed reference materials. USSOCOM will provide training to the components on the programming, operation, and function in this mandated endeavor. Individuals will be required to train other SOF medics at their home station. Staff from this office will also be briefing the SOCs and educating the command on this surveillance process. More to follow!!

EMT-P Bridge Program- The third class for 2002 is in full gear and the course gets better each time. This program will provide the medic only those skills and training opportunities afforded to the civilian Paramedic. We will fight to continue this bridge as long as the community needs it. We are looking at two classes per year.

Joint Medical Enlisted Advisory Counsel- The quarterly JMEAC convened in late July and discussed many topics concerning our SOF medics. At the very top is promotion, SDAP, and education. The wheels of progress move slowly but the issues are being acted upon! Here are the proposed dates of the

upcoming meetings:

- * 6-7 Oct 02, UASOC will host at FT Bragg, NC (this time line coincides with a SOCM graduation on the 8th)
- * 29 Nov 02, USSOCOM will host an Enlisted meeting in Tampa before the SOMA Conference.
- * 25-26 Feb 03, NAVSPECWARCOM will host in San Diego.
- * 27-28 May 03, AFSOC will host at Hurlburt Field.

Medical Equipment- The next JMEAC will be in OCT 02 and will display the medical kit from the SOF community. The object of this approach is to provide a venue where SEMAs can see what kit the units are using. USASOC will display the proposed updated SF Tac Set which will have the newest and greatest kit going.

SOF Paramedic- The quarterly Board of Regents (BOR) meet in late July and all the command surgeon's agreed to recommend that the standard for SOF Enlisted Health Care Provider be "Certified Paramedic". This will overcome the following major hurdles:

- It would relieve the community of the NREMT standards but keep the medics trained at the Paramedic level.
- As a state agency USSOCOM through the JMEAC & BOR can set the mission required standards for their SOF medics.

If you have suggestions, concerns and/or recommendations for the JMEAC, pass them along to your SEMA and it will be addressed. But you have to....

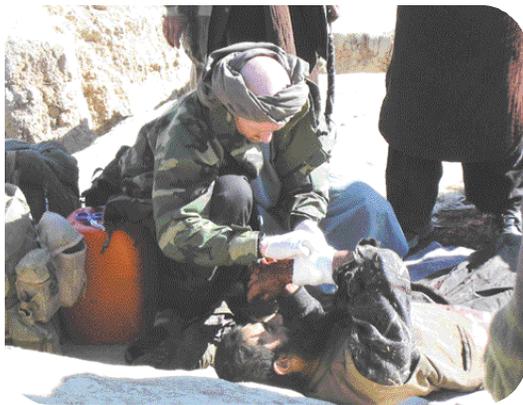
"SEND IT"

All ground is "LEVEL" at the foot of the cross

COVER

This picture depicts a SOF medic giving aid to a POW in Afghanistan

Photos courtesy of USASOC Sine Pari



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The views contained herein are those of the authors and do not necessarily reflect official Department of Defense position. This publication does not supercede any information presented in other Department of Defense publications.

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From The Staff

As we continue to involve you, our readers, in the production of this journal, your submissions and photos are what are needed to make this journal unique. It is a sharing of your missions and your lives as you go forth as instruments of national foreign policy. We can't do it without your input. You are what the journal is all about.

This journal is one of the most excellent and righteous tools we have to span all the SOF services, to share medical information and experience unique to this community. The JSOM survives because of generous but time-consuming contributions sent by clinicians, researchers and former medics from all the services who were SOF qualified and/or who served with SOF units. We need your help. Get published in a peer-review journal NOW. We are always looking for SOF-related articles from current and/or former SOF medical veterans.

If you have contributions great or small... fire 'em our way. Our E-mail is: JSOM@socom.mil.

A recent addition to the JSOM is the offering of CMEs. We are currently working with USUHS, our sponsor for CMEs for the physicians, PAs and nurses. In this edition, you will find CMEs offered on "Heat-Associated Illness and Part One of Going Beyond Thin Air." In this edition of the JSOM, we honor our fallen brother, Chief Petty Officer Matthew J. Bourgeois, killed in support of Operation ENDURING FREEDOM .

Word from the "field" continues to be the same... they would like to see the following types of articles in future JSOMs: Tricks of the Trade...anything from simple more effective bandaging to doing more with less (supplies, meds), keeping IVs warm, treatment of hotspots and blisters, Colloids vs. Crystalloid fluid replacement, IV infusion in extremities vs. intraosseous fluid infusion; Poor-man's Gatorade recipe, improvised laxatives or anti-diarrheals or anything improvised for that matter; herbal medicine...any relevance or uses that are legitimate; articles dealing with trauma, infectious disease processes and/or environment and wilderness medicine type articles; more photos accompanying the articles or alone to be included in the photo gallery associated with medical guys and/or training.

The fact is most everybody that has read an article on a technique or concept knows of another way of doing the same thing that's perhaps faster, easier, or, dare I say...better. Just like any patrol or observation of a target...the more eyes the better. If you, the readers, have knowledge of such things as listed above or at least know where to find info on a particular subject...let us know here. We'll hunt down where you think you saw that information and see if we can't either re-print it for the rest of the readers or at the very least pass along where information of interest can be found. OK, enough said...keep your eyes open and let us know. Thanks.

Lastly, our distribution list continues to expand daily. If you want to continue to receive the JSOM when you PCS, please send us your new address as soon as you know it so we can make the changes in our distribution database. We are losing a lot of money in returned postage; you can help prevent this. Either fill out a change of address form and mail it to us or send it to JSOM@socom.mil. Enjoy this edition of the journal, send us your feedback, and get those article submissions in to us:

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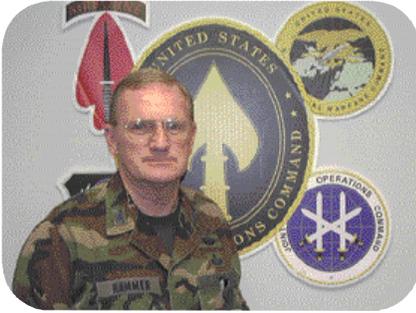
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Colonel Hammer's military and medical career began in 1958 when he served as a U.S. Navy Combat Medical Corpsman attached to U.S. Marine Corps infantry, artillery, and communication/reconnaissance units. Following discharge, he completed his BS and MD degrees at the University of Michigan in 1967 and 1970 respectively. Following nine years of civilian medical practice in a multi-specialty group in Grand Rapids, Michigan, he reentered military service as a Flight Surgeon at Beale AFB. In 1984, he completed the Air Force Residency in Aerospace Medicine at Brooks AFB, Texas, during which period he earned a Masters in Public Health Degree from

Harvard University. Colonel Hammer has spent the majority of his career in aerospace medicine and direct line support assignments, has commanded three medical groups, and has been assigned to the ARRS/SG,

MANAGING EDITOR

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CPT Anderson enlisted in the Army in 1980. Upon completion of the Combat Medic course, volunteered for Airborne and Special Forces training. Assignments encompassing 13 years as a SF medic include: Team medic- C/3/10th SFG(A), Instructor at Med Lab- Ft Bragg, Medic- 1st SFOD-D. CPT Anderson was accepted to the Military Physician Assistant program and subsequently commissioned in 1995. Assignments from that time to present include: 1/9th INF Regiment, 2/504th PIR, 82d Abn DIV, 2/7th SFG(A), and currently assigned to the USSOCOM Surgeons Office as the Command PA. Education and qualifications include: B.S. Southern Illinois University-'79, B.S. University. of Oklahoma-'95, and MPAS University. of Nebraska-'97. Jump Master, SERE, HALO, Combat Diver, Dive Medical Technician, Flight Surgeon, Dive

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Maj DuGuay joined the Army Reserve in 1987 and served as a nurse in a Combat Support Hospital unit for three years before switching services in 1990 to become an Air Force C-130 Flight Nurse. She is currently an IMA reservist attached to the SOCOM/SG office. Maj DuGuay has a Bachelor's in Nursing and and a Masters in Business Administration / Management. Her career includes being a flight nurse in both the military and private sector, 15 years of critical care and emergency room nursing experience, an EMT and a legal nurse consultant. She also serves as the military liaison to her Disaster Medical Assistance Team (DMAT). Prior to the SG office, Maj DuGuay's experience at USSOCOM includes an assignment in the Center for Force Structure, Resources, Requirements, and Strategic Assessments.

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6. Remember that your audience is inter-service, civilian, and international.
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12. Again, the JSOM is your journal. It is a unique chance for you to pass your legacy to the SOF medical community.

Take advantage of the opportunity.



USASOC



Rocky Farr, MD
COL, USA
Command Surgeon

As I have been getting approval and funding (a special thank you from us all to Major Hank Sully) for the fielding plan for new medical equipment sets (MES), the first of which get to an operational special forces group next month, I have seen may new items. At the risk of violently upsetting all, or most all, orthopedic surgeons, I have added an external fixator set to one of the MES chests. The same external fixator set that is found in an Army forward surgical team MES.

We in Special Forces are swinging back toward our true core mission of unconventional warfare (UW). UW means longer evacuation routes, sometimes no evacuation routes, improvisation, and sometime it means different care for different wounded, depending on the tactical situation and importance. It also means forward surgery, either from a Forward Surgical Team brought further forward than they are used to, an allied surgical element, an indigenous surgeon, or a Special Forces surgeon or medic. In many cases that surgeon will also be the shooter and the post-op nurse.

Do I think Special Forces medics and Navy SEAL Corpsman can master the skills of how to perform external fixation? Damn right I do! Colonel Keenan has already done it at the Joint Special Operations Medical Training Center (JSOMTC). Colonel Frank Anders, a former 18E, now orthopedic surgeon has brought this to the JSOMTC, made

a great instructional videotape showing the procedure to JSOMTC instructors, both Navy SEAL and Army 18D, and has *rods will travel* for interested groups.

Who needs this new technology? Open, compound fractures are quite common wounds. Until now the only thing an 18D could do would be a windowed cast. We all, by now, have heard the stories of the difficulties of nursing care in the guerrilla warfare operational area. These wounds, managed in this way (the World War II way) are tough to care for.

If I were in mountainous county with a femur fracture facing a long donkey ride I would sure vote for external fixation rather than casting for that bumpy trip. If the wounded guerrilla chief were going to be kept alive to rally the troops, I would want it (and he'd get damn good nursing care too!).

I now turn the rest of the space over to Colonel Anders:



Traditionally, 70-80% of survivable combat wounds have been in the extremities. That percentage is increasing with the use of body armor. Salvage of limbs with high and low energy wounds but good distal neurovascular function is greatly dependent on the early management of those wounds. The treatment of compound (open) long bone fractures is difficult even under optimum conditions in a trauma. This difficulty is multiplied many-fold in the austere environment of the Special Operations Forces (SOF) Medic. Immobilization, especially in comminuted fractures, is almost impossible for the femur, tibia or humerus with casting or splinting techniques. Skeletal traction is only appropriate when the patient can remain at bed rest for prolonged periods.

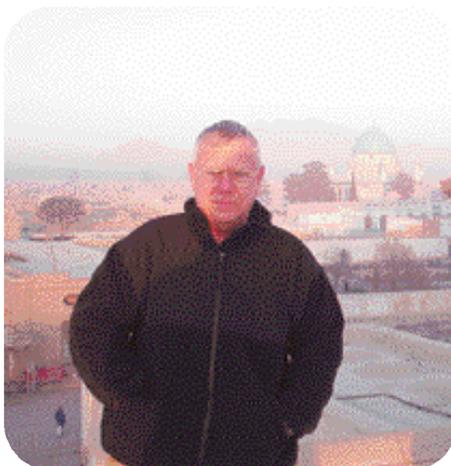
The greatest hazard in transporting or repositioning a patient with such an injury is nosocomial damage to the adjacent neurovascular structures by sharp bone fragments. Patients with these complex injuries treated by SOF Medics are moved frequently and by field expedient methods in their areas of operation due to changing battle conditions and the proximity of hostile forces. In addition, Special Operation missions often require deep insertion of teams into hostile environments and denied areas where timely airborne CASEVAC is impractical or impossible. This has been emphasized in debriefings

by many of our SOF Medics who returned to JSOMTC from active theatre operations for SOFMSSP.

I have effectively immobilized and provided long-term treatment of such high-energy fractures by external fixation in more than one hundred (100) cases during twenty years of orthopedic surgical practice. The techniques are set forth and validated in articles by French, Tornetta and many other authors as well.

Knowledge of and attention to cross sectional anatomy of the extremity is essential for safe placement of external fixation pins to avoid damage to the neurovascular structures by the pins. Tests here at JSOMTC demonstrate that our SOF Medics are proficient in these areas. I was able to verbally direct seven SOF Medics through successful placement of the Hoffman II external fixation device without difficulty, demonstrating that only basic mechanical skills are required for proper pin insertion and assembly of the device.

We expect that the external fixation device will be a useful new tool in the SOF operator's toolkit. Just as you can't fix every problem with a hammer, so the external fixator will not solve every orthopedic problem. However, when properly applied under the appropriately indicated conditions, this device will save SOF limbs and even lives.





NAVSPECWARCOM



Larry Garsha, MD
CAPT, USN
Command Surgeon

It is the time of transition during the summer and many of us are moving to new duty assignments. Here at NAVSPECWARCOM we have sadly waved farewell to Master Chief Cavolt. He has made a tremendous contribution to NSW over the years and his tenure at the head shed leaves very large footprints. He will be heading to Logistic Supply Unit TWO. Master Chief David Jefferson is our new Force Master Chief Corpsman. He comes from the training department of SEAL Team THREE.

We have a new Commander, RADM Albert Calland. He comes from OEF where he led northern base as SOC Commander for CINCCENT. He brings the perspective of a war fighter from the front. RADM Olson is transitioning to Chief of Naval Operations office in charge of planning.

Two new physicians have been added to NAVSPECWARCEN. Lieutenants Vergilio and Minarcik have joined Lieutenants Ware and Ambach. Lieutenant Lieske is joining the staff at Logistics and Supply Command TWO. Chief Belanger is the new senior enlisted at Group Four, and Chief Moore is the new senior enlisted at Group ONE. Welcome aboard one and all!

Master Chief Jefferson held the NSW Medical Enlisted Advisory Committee. One of the many issues discussed including adding NEC 8404 Corpsmen to the Special Warfare combat Crewman boat crews. These individuals will be able to attend

JSOMTC for special medical training per detailing availability. Because of anticipated shortfalls in the corpsman community renewed vigor will drive recruitment. Other services beware we are on the prowl.

Other projects on deck and at bat includes recent instructions on Supplement Use in Navy Special Warfare, Skin Cancer Prevention, and Naval Special Warfare Health Promotion and Protection. Presently in the chop chain, look for them to be finalized in October. Pre and post deployment screening is another issue demanding attention of deploying forces. SOCOM will be hosting a "train the trainer" class for handheld units for deploying forces. Finally, we have launched beta testing of the NSW-MD. The Naval Special Warfare Medical Database will include capability to print daily binnacle lists as well as track immunizations, daily complaints, even medical limited duty.

This should be ready for promulgation force wide by December.

Work is continuing on new platforms to fight from. The ASDS is completing sea trials on schedule and the SSGN is starting to be planned by the submarine community.

These "Modern Marvels" will maintain our SOF position in the littoral and surf zone.

The new tourniquets are on the way and so is a new clotting bandage for deep wound hemorrhage.

Anyone out there with new ideas should contact Master Chief Jefferson @ 619-437-5150.



AFSOC



James Dougherty, MD
Col, USAF
Command Surgeon

ALTERNATIVE MEDICINES, SUPPLEMENTS AND PERFORMANCE ENHANCERS

The Journal of the American Medical Association had a recent article about people taking the herbal anti-depressant, St. John's Wort. If you are a cancer patient, on chemotherapy, you don't want to take this "alternative" agent. The researchers found that cancer therapy is 40% less effective while taking this herbal. Well, this is about the sixth herbal in the last couple of years that we have found to have some decidedly negative aspects. From contaminated herbals causing urethral cancer, to herbals that alter the effects of blood thinners, it appears that these "natural" compounds influence the body in complex ways, especially in combination with pharmaceuticals. This is a great disappointment because many people are convinced that because a chemical or herb is "natural" then it is always good for you.

We don't have a lot of research on this stuff, but one thing I DO know is that the little data we have shows that the side effects and adverse reactions are sometimes significant: sometimes only annoying, but occasionally deadly.

Over the last few years doctors have been repeatedly shocked by how often their patients use supplements and herbals: in some surveys 40% are taking something they don't tell their doctors about. People are taking an astonishing number of pills and elixirs every day. I often tell a patient to bring in all their medications, and sometimes the response is, "all of them?" You can bet that they will bring a bag of vitamins, herbs, and other power aids. What's scarier is the fanciful explanation that justifies this

use, always referencing market ads, or articles in non-peer reviewed magazines, some buddy's testimony. The complex and elaborate reasons why people take these things would be funny-- if only it was harmless.

The docs share the blame for this situation with other responsible parties. The medical profession is WAY behind on knowledge about alternative agents, and as we scramble to learn more, there have been some sobering lessons. But complicating our efforts to get smarter is the fact that Congress decided that these compounds are exempt from regulation in the way that standard medicines are regulated: new formulations pop up daily, have their day in the sun, and are discarded-or worse, their names are just changed to give the impression of being new (and to dissociate themselves from any bad connotations of the old name).

This puts potentially harmful agents in the hands of people who have no recourse to reliable sources of information. I have nightmares about patients with real medical problems, or chronic injuries, who are desperate for improvement, but have to wade through the snake oil to get to something that can help them.

Setting aside some specific and very potent chemicals, such as steroids or amphetamines, which are prohibited, the remaining plethora of products have in general never been shown reproducibly to be able to increase strength, stamina, energy level, or alertness. (I know about the Rangers and creatine: it aint worth the effort). Each product has its fervent adherents, but where scientific data is available, the evidence is usually lacking that the claims made for

the product are true. In most cases, a critical look at the ingredients will show that the constituents of the product are vitamins or food building blocks that we get in large enough amounts in our diet already. Amino acids are a real favorite.

For these reasons USSOCOM policy prohibits the use of so-called "performance enhancers". Air Force flying personnel, operational duty personnel, and PRP personnel must also notify their flight surgeon of any use of supplements. It needs to be put in the medical record so that any potentially

unsafe circumstances can be avoided. There's a brochure online about this and other supplements at <http://www.brooks.af.mil/web/af/altmed/HOME-FRAME.htm>.

I recommend a skeptical review, and caution, before taking any of these products. Also, consult with reliable sources, and avoid prohibited items.

Lastly, very few of us are interested in the male breast enlargement that has recently been reported from taking St. John's Wort.





SOF Med Plans/Ops Update

LTC Lou Nelson

Chief, Medical Operations

Our nation's response to the attack on our country on 9-11 was both fast and far-reaching. I'd like to briefly discuss what has occurred in the SOF Medical Plans and Operations arena since 9-11.

The initial introduction of SOF into an isolated *Forward Operating Base* (FOB), far from coalition or host nation medical facilities capable of meeting our needs, forced SOF medical planners to be very deliberate when determining the initial SOF medical package. Also, the limited availability of lift and the requirement to get as many SOF operators as possible on the ground first forced us to maintain the smallest possible medical footprint. This, by the way, will continue to be a valid constraint for SOF medical planners in future operations.

The USSOCOM SG's staff provided functional expertise to the USCENTCOM SG as required and a very capable medical package was pushed into the *Time-Phased Force Deployment Data* (TPFDD) early on. The overall goal was to provide responsive CASEVAC forward of the FOB and adequate Level II surgical/resuscitative capability, very limited Patient Hold capability, and a robust forward basing of conventional AE capability at the FOB. The Level II capability was initially provided by an AF *Mini Forward Surgical Team* (MFST) and later by an Army *Forward Surgical Team* (FST.) Patient Hold was initially provided by a AF *Mobile Aeromedical Staging Facility* (MASF) with *Critical Care Aeromedical Transportation Team* (CCATT) augmentation. An *Aeromedical Evacuation Liaison Team* (AELT) with a few AE Crews and CCATTs ensured our ability to quickly evacuate stabilized patients out of the FOB on opportune conventional aircraft as required.

This basic medical capability package proved to be very adequate and can be put together at a cost of less than 50 personnel spaces. Often a defensible request from the SOF medical planner to the Commander to support a large SOF operation in a truly austere environment with long evacuation lines to the next level of medical care. This capability was repeated at subsequent SOF FOBs and

proved our ability to meet SOF medical requirements with true Joint medical packages.

SOF received Level II surgical/resuscitative capability from all three services. The AF met this need with the MFST and *Expeditionary Medical System* (EMEDS) package, the Army with the FST, and the Marines/Navy with the *Shock Trauma Platoon* (STP) with Fleet Surgical Team augmentation. The AF met our limited patient hold capability requirement non-doctrinally with a MASF with CCATT augmentation and a specific agreement with TRANSCOM that they would be prepared to hold patients for up to 24 hours. In other locations, this capability was provided by an Army Medical Company (-). AE assets including AELTs/ELTs, CCATTs and AE Crews were provided as far forward as conventional aircraft operated. This use of opportune conventional aircraft saved our limited CASEVAC assets to support SOF operations forward of the FOB.

Overall, this basic medical model is now tested/ proven and will likely be repeated to support future operations. However, this has worked well for two reasons. First and foremost, the medical successes of this operation all started with the amazing dedication and competence of the SOF medic as the initial responder. The tremendous skill and determination of our SOF medics, whether out with their teams conducting their SOF mission or standing ready to support our CASEVAC requirement, is and always will be the key and to meeting SOF combat medical requirements. Second, SOF has received and will continue to require the support of conventional medical and aeromedical assets to adequately meet overall SOF medical requirements. Although very capable, the overall amount of organic medical capability within SOF is very limited. Effective integration into the conventional medical system, as soon as possible, will continue to be critical.

The real challenge to us as SOF medical planners will be to maintain our ability to meet overall SOF medical requirements as the "Global War on Terrorism" continues. The potential for even greater

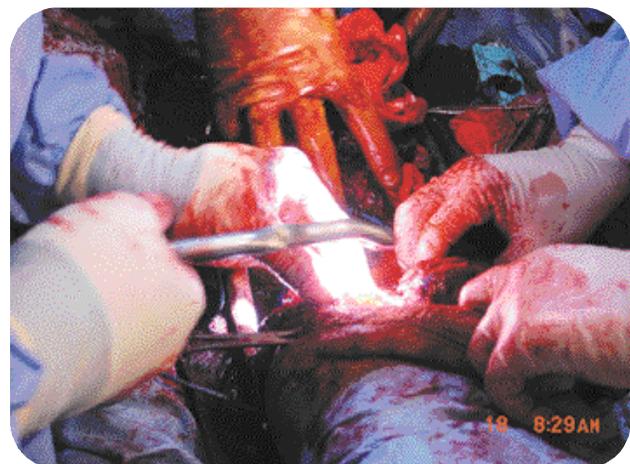
SOF involvement in other Combatant Commander areas and the realization of our limited organic assets will make the "ART" of SOF medical planning even more crucial. Its not enough to just look up all of these acronyms, we must learn the true capability and limitations of these medical assets.

We must recognize what works well and admit what does not, forget service rivalries, learn to identify and articulate specific capability requirements, and provide the most capable JOINT medical support system possible to support our most critical SOF asset, OUR PEOPLE!



An inside view of a FST
*Photo courtesy of LTC Lou Nelson, Chief of
Medical Operations, HQ USSOCOM*

FST treats trauma injuries related to landmine blasts





USSOCOM Biomedical Initiatives Steering Committee (BISC)

Robert Clayton

The Biomedical Initiatives Steering Committee (BISC) conducted the third quarter meeting at Fort Bragg, NC 16-17 August 2002. As I have stated before the BISC was formed to support the medical research and development efforts for Special Operations. The Component Surgeons or their appointed representatives meet to focus on medical issues that impact on the mission, training and performance of SOF. Primary research areas are: Diving Related Studies, Performance Enhancements, Combat Casualty Care, Medical Informatics and Graduate Research Studies. The BISC is regulated by USSOCOM Directive 40-1.

The BISC uses Major Force Program-11 funds to support these studies. There are a lot of medical issues that are not SOF specific but because SOF is the "Tip of the Spear", the situation creates a SOF specific need. One of the methods that the BISC employs is to leverage minute amounts of funds against the Services Medical R&D community. This strategy not only accelerates the development of several programs that are important to our mission but also provides a bigger return on our investment.

Each SOF Component has a BISC representative. The function of these representatives is to review their service requirements and to provide the BISC with Task Statements for research topics. A Task Statement is usually a two page description of the need, related background and a desired solution. On an average the BISC supports 10 to 12 Task Statements per Fiscal Year (FY). Once the Task Statements are written and accepted by the BISC, the USSOCOM MEDTECH Program Manager (PM) sends out request for pre-proposals. Once the pre-proposals are received they are reviewed, screened and if applicable presented to the BISC for review and approval. Once the BISC approves the pre-proposal, the PM requests that the organization submit a full proposal with any clarifications that the BISC may have noted. The full proposal is again reviewed

and is then put before the BISC, which recommends a funding prioritization. The BISC accepts out of cycle Task Statements and unsolicited proposals. Depending on the topic, applicability and status of funding these may be held for future selection.

THE BISC MEMBERS:

USSOCOM, Col. David Hammer, Chairman
USASOC, COL. Warner (Rocky) Farr, voting member
AFSOC, Col. Matthew Coatsworth, voting member
NAVSPECWARCOM, CAPT. Frank. Butler, voting member
JSOC, COL. Steven Swann, voting member
JSOMTC, COL. Kevin Keenan, advisor non voting member.

FY02 RESEARCH IS CURRENTLY UNDERWAY ON THE FOLLOWING PROJECTS:

1. IMPACT OF VARIOUS BREATHING GAS MIXTURES EFFECTS ON DCS
2. ASSOCIATED WITH AC-130H/CV-22) 2000-1 MOTION SICKNESS PREVENTION IN NSW--AN EXAMINATION OF AIRSICKNESS PREVENTION IN SPECIAL OPERATIONS FORCES
3. BIOMEDICAL R&D INFORMATION PROJECT
4. SOF COMMITTEE ON TACTICAL COMBAT CASUALTY CARE (TCCC)
5. BIOMEDICAL TRAUMA EDUCATION SERIES

THE FOLLOWING PROJECTS ARE BEING CONSIDERED FOR FY03:

1. MECHANISMS OF INJURY IN REFRACTORY DCS AND AGE
2. MAXIMUM BREATH-HOLD DIVE DURATION STUDIES
3. FULL-FACE MASK PURGING FOR THE MK 25 UBA
4. MEDICAL SUPPORT OF HIGH-SPEED BOAT SHOCK MITIGATION STUDIES
5. STRESS FRACTURES IN BUD/S TRAINING

- 6. COMPUTER ASSISTED THERMAL PROTECTION PLANNING IN SOF
- 7. EFFECTS OF LOW GRADE HYPOXIA AT NIGHT IN SOF AIRCRAFT OPERATIONS

MEDICAL R&D UPDATE

The *Hemostatic Dressing (HD)* has gone to the Institutional Review board at the American Red Cross, in-turn it will be reviewed by the Human Subjects Research Review Board at the Medical Research and Materiel Command prior to being submitted to the FDA for approval of the Investigational New Drug protocols. Once the FDA grants approval SOF Component Surgeons will begin administering the informed consent and training in order to be allowed to use the HD. The first lot of HD will start coming off of the assembly line in mid August, once the efficacy of the first lot is confirmed they will be produced and distributed to deployed SOF elements.

The *one handed tourniquet* has been through the preliminary design and configuration test with

distribution occurring in early August 2002. SOF Components will be distributing them with instructions on how to use them.

The *Chitosan Dressing* has undergone efficacy trials and is a very promising replacement for the HD. Indications are that the Chitosan dressing will be approved by the FDA for external use in the September/October 2002 timeframe.

Hemoglobin Based Oxygen Carriers (HBOC) is on the horizon. A Mission Needs Statement is being developed to try and ramp up the research in support of SOF requirements.

The *CD-ROM version of the SOF Medical Handbook* has been distributed. It did not turn out as previously planned with hyperlinks to the SOF Medical Diagnostics Program and an embedded electronic book set, once funds can be allocated this maybe possible.

The Special Operations Computer Assisted Medical Reference System (SOCAMRS) 2002 should be ready for distribution in early October 2002.

In future editions of the journal you will start seeing some of the reports from the studies that the BISC has funded.



Heat Associated Illness

John W. Gardner and John A. Kark

ABSTRACT

Heat associated illness or injuries as commonly known to us in the military, presents itself in a variety of signs, symptoms and system involvement. Heat injuries and their consequences can range from just being uncomfortable to coma and death. Because of their potential hazard in any environment a good review and continuous vigilance is paramount. This paper reviews the pathophysiology, risks, and syndromes involved with subsequent treatment options.

OBJECTIVES

1. Explain the pathophysiology and physiological changes that occur in the body during the acclimatization process.
2. Describe the signs, symptoms and treatment of heat-associated illness as suggested by the authors.
3. Understand the differentiation between the types of heat illness and their treatment.

Complete test on page 52--Answer sheet on page 56

Completion of this article and test offers 1.25 CME and 1.4 CNE/CEH

EDITORS NOTE: This article was previously printed in Strickland: Hunter's Tropical Medicine and Emerging Infectious Diseases, 8th Edition, page 140

DISCLOSURE: The presenters have indicated that, within the past two years, they have had no significant financial relationship with a commercial entity whose products/services are related to the subject matter of the topic they will be addressing or a commercial supporter of this educational activity.

DEFINITION. Heat illness encompasses a spectrum of metabolic disorders deriving from the combined stresses of heat exposure, exertion, and thermoregulation. These include classic (environmental) and exertional heat stroke, heat exhaustion, heat injury, dehydration, heat cramps, acute renal failure, hyponatremia, and rhabdomyolysis. In addition, there are some minor heat-associated illnesses, such as heat rash, heat edema, parade syncope, and sunburn. Heat illness represents primarily a continuum of multisystem illness related to elevation of core

body temperature and the metabolic and circulatory processes (including changes in fluid and electrolyte balance) that are brought about by heat exposure, exercise, and the body's thermoregulatory response.

PATHOPHYSIOLOGY

The physiologic responses to heat stress in humans are controlled by a sensitive and efficient thermoregulatory system. Optimal body temperature is maintained through a balance of environmental temperature, endogenous production of heat, and effective loss of body heat. In hot environments

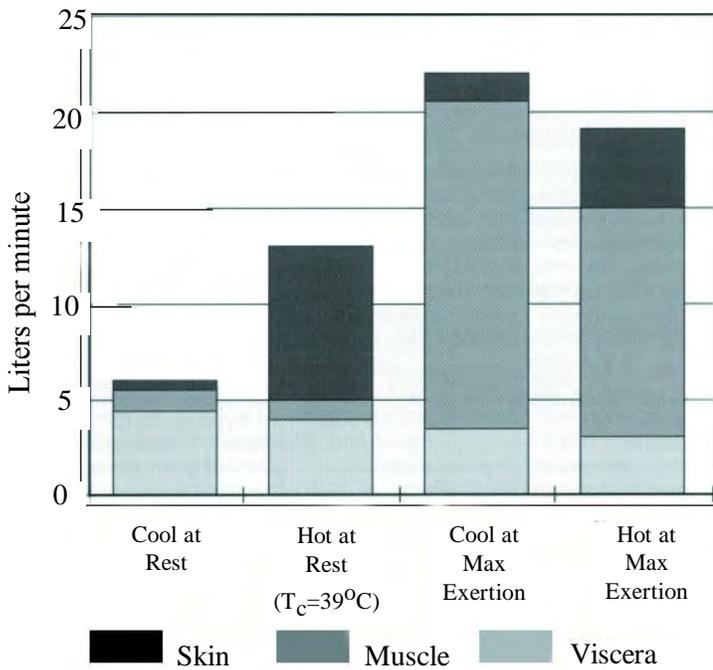


Figure 17-1. Estimated distribution of blood flow to the muscle, skin, and viscera as fraction of cardiac output at rest and with maximum exertion under cool and hot conditions. (Adapted from Rowell LB: Human cardiovascular adjustments to exercise and thermal stress. *Physiology Review* 54:75, 1974.)

thermoregulation is accomplished primarily by redistribution of blood flow to carry internal thermal energy to the skin, where heat dissipation occurs through conduction, convection, radiation, and evaporation. *Figure 17-1* shows the estimated distribution of blood flow to the muscle, skin, and viscera as a fraction of cardiac output at rest and with maximum exertion under cool and hot conditions. A large temperature differential between the skin and its surroundings provides efficient heat dissipation under cool conditions, requiring only about 5% of resting cardiac output going to the skin. However, under hot conditions there is inefficient heat dissipation and blood flow to the skin increases dramatically even at rest, requiring a large increase in cardiac output (shown when rectal temperature has increased to 102°F [39°C], implying maximum thermoregulatory effort). This contributes to the fatigue one may feel even at rest in the heat.

ENVIRONMENTAL EFFECTS

Optimal thermal balance can work effectively within certain limits. Wind, solar radiation, and humidity play important roles in the efficiency of heat dissipation through convection, radiation, and evaporation. Even at rest in cool environments the

body loses about 2 L of fluid per day through respiration and perspiration; this may be imperceptible in dry or windy weather conditions. As more body heat must be dissipated, these fluid losses increase due to heavier sweating, which may exceed 2 L/hour (up to a maximum of 12 to 15 L/ day). Heat losses from the body to the environment through radiation, conduction, and convection are markedly reduced when the ambient temperatures rise above 86°F [30°C]. Under such circumstances, sweating becomes the most important means of heat loss. Evaporation is severely hampered by high ambient humidity. Thus, the combination of high ambient temperatures and high relative humidity sets the stage for the development of heat illness.

IMPACT OF EXERCISE

With strenuous exercise there is a three- to six-fold increase in cardiac output due to increased blood flow to exercising muscle (*Fig. 17-1*). Exercise generates large amounts of heat and elevates body temperature, since about three fourths of the metabolic energy used by muscle during exercise is converted into heat within the body. As body temperature elevates, more blood flows to the skin for heat dissipation. Under cool conditions there is efficient heat dissipation, and the amount of blood flow to the skin increases only marginally. However, under hot conditions there is less efficient heat dissipation and a larger amount of blood flow goes to the skin. This produces peripheral pooling of blood, which decreases total cardiac output and further limits blood flow to muscle, thus reducing peak exercise performance in hot environments. These circulatory demands of sustained exercise and heat stress may also encroach on visceral blood flow to the extent of producing organ dysfunction or cellular injury; e.g., watery or bloody diarrhea often seen in marathon runners, and perhaps acute renal failure and encephalopathy often seen in heat stroke.

ACCLIMATIZATION

Heat exposure and/ or regular strenuous exercise, which raises core temperature and provokes heat-loss responses, produces heat acclimatization that improves the body's response to heat stress within a few days. The acclimatization achieved is a process of changes in circulation, sweating, and sodium and water balance, as well as

a gradual increase in metabolic efficiency of energy utilization. Most of the physiologic improvement in heat tolerance occurs within 10 days of combined heat exposure and regular exercise. In acclimatized individuals blood volume increases, the heart rate is lower due to increased stroke volume, sweating begins earlier with a higher sweat rate and low sodium content, and the threshold for cutaneous vasodilatation is reduced. These changes improve transfer of body heat from the core to the skin and enhance heat dissipation at the skin. Although sodium is conserved with heat acclimatization, the need for water replacement is not reduced because sweat volume increases.

Water losses in exercising individuals are universal, but salt loss is excessive primarily in unacclimatized individuals. Thus, hypertonic fluid losses occur primarily during the first two weeks of training in a hot environment. Acclimatized individuals excrete large volumes of sweat with minimal salt content, and hypotonic fluid losses predominate. Dehydration may thus be due to isotonic, hypotonic, or hypertonic fluid loss.

EPIDEMIOLOGY AND PREVENTION

Numerous risk factors have been identified for heat illness, which relate primarily to weather and training circumstances; clothing and shade; acclimatization; physical fitness and body type; illness, medications, and alcohol; water and salt; and personal education. These factors are directly related to the occurrence and severity of heat illness, and attention to them can greatly reduce its morbidity and mortality and minimize the impact of heat stress exposure.

WEATHER AND TRAINING CIRCUMSTANCES

Temperature, humidity, and solar and wind conditions greatly influence the body's ability to maintain thermal balance. The wet bulb globe temperature index ($WBGT = 0.7 \times \text{wet bulb temperature} + 0.2 \times \text{black globe temperature} + 0.1 \times \text{dry bulb temperature}$) was developed as a weather index that is linearly related to sweat rates during moderate exercise. It has therefore been adopted as a practical index of risk for heat illness during exercise in hot weather conditions. Exertional heat illness rates can be minimized by progressively limiting the level of exercise, increasing rest periods, increasing hydration, and reducing heat-retaining clothing as the WBGT rises above 70°F [21°C]. These modifications are implemented in a standard manner in U.S.

military training for four categories of high WBGT (green flag, 80° to 85°F [26.7 to 29.4°C]; yellow flag, 85° to 88°F [29.4° to 31.1°C]; red flag, 88° to 90°F [31.1° to 32.2°C]; black flag, 90°F [32.2°C] or higher). As a rough guide, regulations allow exercise to continue with caution during green flag, strenuous exercise (e.g., "marching at standard cadence") is suspended for unacclimatized individuals during yellow flag, strenuous exercise is curtailed for all individuals during red flag, and all outdoor physical training is suspended during black flag conditions. These guidelines were designed to minimize the risk during marching (at a metabolic rate about six times basal). An adjustment is necessary to protect individuals who are running (at 12 to 14 times the basal metabolic rate); under these circumstances risk for exertional heat illness increases dramatically as the WBGT rises above 65°F [18.3°C]. Restrictions are generally recommended at lower WBGT levels in athletic programs, and detailed recommendations have also been made for categories of industrial work, including criteria at which work should stop (see Bell and Watts in Bibliography).

CLOTHING AND SHADE

In mild to moderately severe weather, minimizing heat retention by clothing is an important benefit. Light, loose-fitting clothing with head cover or umbrella protects from sunburn, the radiant heat of the sun, and allows air circulation to enhance evaporation of sweat. During extreme heat with sun exposure, cover by loose-fitting clothing also reduces heat uptake and permits tolerance of a longer work period. Provision of shade and air movement is important. One large tree may cool as much as five 10,000 BTU air conditioners. An example is the provision of shade by planting trees in the mountainous area of Arafat in Saudi Arabia, where about a million pilgrims gather annually in a 10km² area. Individuals required to perform heavy exertion in the heat will have much lower risk for heat illness if they spend most of their time in a cool environment, ideally with shade, ventilation, and low humidity, as may be provided by reflective shielding, heat insulation, or air conditioning. Other methods of providing a cool individual environment include cooling vests and suits used in special industrial operations.

TRAINING AND ACCLIMATIZATION

The physiologic changes of acclimatization increase the capacity for and efficiency of heat dissipation.

pation. Due to increased sweating, water intake requirements go up. There is no hot weather training that decreases water requirements. A sensible program for rapid acclimatization to heavy work in a hot climate is to perform at about half-maximal effort for 2 hours per day while resting in a cool place otherwise. This should

be accompanied by generous hydration and a temporary increase of salt in the diet (as salty food, not salt tablets). Although the benefits of acclimatization can be seen within 2 to 4 days, full acclimatization requires 2 to 4 weeks.

PHYSICAL FITNESS AND BODY TYPE

Poor physical fitness and obesity are interacting risk factors for exertional heat illness. In male U.S. military recruits beginning basic training, those in the slowest quartile of run-time had a 5.6-fold higher risk for heat illness than those in the fastest quartile. Those who were in the highest quartile of body mass index (BMI) had 3.6-fold higher risk for heat illness than those in the lowest quartile. Morbid obesity provides even higher risk for heat illness, and individuals with marginal cardiac reserve are particularly susceptible.

ILLNESS, MEDICATIONS, AND ALCOHOL

Chronic illness is a primary predisposing factor, and the epidemics of heat-related deaths (often several hundred) seen during heat waves are due primarily to classic heat stroke in individuals with marginal cardiac reserve who are unable to maintain cool surroundings and / or adequate hydration. The largest group at high risk is elderly persons of low socioeconomic status. Certain medical conditions and medications can put an individual at high risk for developing heat illness. These can make adjustment to hot environments more difficult, and include acute illness, especially infections and febrile conditions, chronic illness, prior history of heat

TABLE 17-1. Drugs That Interfere With Thermoregulatory Mechanisms

Drug Class-Examples	Physiologic Effect
Diuretics--furosemide, thiazides, caffeine	Water and electrolyte loss
Antihistamines--chlorpheniramine	Suppress sweating
Anticholinergics--atropine	Suppress sweating
Antiparkinsonian-- procyclidine	Suppress sweating
Phenothiazines--chlorpromazine	Suppress sweating, anhidrosis
Tricyclics--tranylcypromine	Suppress sweating, hyperpyrexia
Butyrophenones--haloperidol	Suppress sweating, thirst suppression
Monoamine oxidase inhibitors	Suppress sweating
Sympathomimetics--amphetamine	Suppress sweating, hyperpyrexia
Nicotine	Inhibit vasomotor control
Antihypertensives--propranolol, methyldopa, guanethidine	Inhibit vasomotor control
Thyroid hormones--thyroxine	Hyperpyrexia
Hallucinogens--LSD	Hyperpyrexia
Salicylates, barbiturates	Hyperpyrexia
General anesthetics--halothane	Hyperpyrexia
Alcohol	Unknown

LSD, lysergic acid diethylamide.

stroke, pregnancy, obesity, skin disorders, sunburn, and uncleanliness. Medications, alcohol, caffeine, nicotine, loss of sleep, large meals, and missed meals can also predispose to heat illness and may nullify the effects of acclimatization. Drugs that interfere with thermoregulatory mechanisms are listed in *Table 17-1*. Individuals with diffuse skin disease and anhidrosis (*Fig. 17-2*) are particularly susceptible to severe heat illness. They may not be able to perform even a small amount of work in the heat without inducing heat stroke.

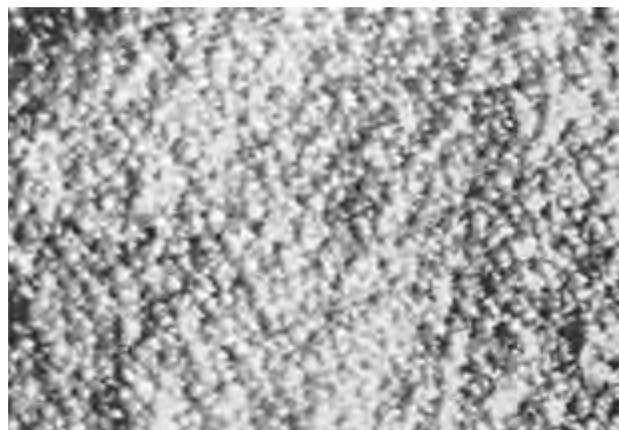


Figure 17-2. An enlarged picture of human skin showing miliaria profunda. Notice the goose flesh-like appearance. (From *Home GO, Mole RH: Trans R Society of Tropical Medicine and Hygiene 44:465, 1951. Reprinted by permission*)

WATER AND SALT

Prevention of heat illness requires proper provision for water and salt requirements. Water

must be available and palatable, and water intake must be monitored. Water requirements are not reduced by any form of training or acclimatization. Tolerance to dehydration cannot be developed, and water supplies are as important for survival as are food and shelter. For full-time outdoor activity in a hot environment, water intake should be about 1 L / hour. For planning purposes, 16 to 24 L of potable water per person per day are needed for drinking and cooking, and at least another 16 to 24 L for washing and bathing. Thirst is not an adequate guide to water needs because it starts only after losses of about 1 L occur. Urine volume and color, body weight changes, and orthostatic blood pressure changes can all be used as guides to adequate hydration. The advantages of carbohydrate / electrolyte beverages beyond their palatability are controversial. High sugar solutions may impede water absorption. Salt losses should be made up (during the first 2 weeks in a hot environment) with a high salt diet, not with salt tablets. *Replace water losses hour-by-hour, and salt losses day by day.* Sustained water replacement much beyond 1 L/hour may result in symptomatic hyponatremia, particularly if heat illness has already affected renal function.

PERSONAL EDUCATION

Each individual must be fully aware of heat illness, its signs, and its prevention. Personal awareness and use of common sense will avoid most problems. Individuals should understand the early symptoms and signs of heat illness, and increased susceptibility with use of certain medications (including alcohol) and during and after recovery from acute (even minor) illness. It is important to avoid excessive fatigue, get adequate sleep, maintain calories and adequate salt in the diet, and avoid alcohol, nicotine, and caffeine. Each individual must maintain adequate water intake, which requires drinking when not thirsty and monitoring urine volume and color, weight changes, and so on. The individual must be aware of the need to hydrate ahead of thirst and before and during exercise. Individuals should wear appropriate clothing, seek shade and cool environments, utilize rest periods, and adjust the intensity and duration of exercise according to environmental conditions and their own sense of well-being. Buddies can recognize the symptoms and signs in each other, assisting in early recognition and management.

HEAT ILLNESS SYNDROMES

The spectrum of major heat illness represents a continuum of multisystem illness, which may be divided into three main levels. The most severe are heat stroke (characterized by encephalopathy), rhabdomyolysis (characterized by muscle necrosis, with liver necrosis as a variant), and acute renal failure. The milder syndromes are categorized as heat exhaustion, and those with intermediate severity are called heat injury. Authors differ on the criteria used to describe each syndrome. Differentiation of heat illness syndromes by rectal temperature is popular in medical texts and emergency rooms, but this is unreliable for assessing severity of heat illness.

Classic (environmental) heat stroke is generally associated with extensive exposure to a hot environment in the absence of strenuous exercise, and is a different illness from exertional heat stroke. It has been associated with anhidrosis (absence of sweating), but anhidrosis is not a consistent finding in exertional heat stroke and consequently is not a useful criterion for categorizing heat illness. Classification of the continuum of heat illness must be made on the basis of neurologic symptoms and evidence of organ damage or dysfunction. The neurologic symptoms in heat illness are those of a generalized encephalopathy, which range from lethargy, confusion, and disorientation to delirium, obtundation, and coma. The organ damage or dysfunction in heat illness is usually manifest as dehydration, electrolyte disturbances, metabolic acidosis, acute renal failure, and/or muscle or liver necrosis. It is valuable to provide an accurate assessment of each patient for encephalopathy, dehydration, metabolic acidosis, electrolyte disturbance, renal failure, tissue necrosis, and in severe disease, disseminated intravascular coagulation (DIC).

HEAT STROKE

Heat stroke is a life-threatening, progressive multisystem disorder reflecting collapse of the thermoregulatory system accompanied by severe neurologic symptoms with or without organ damage. The severe neurologic symptoms of heat stroke include delirium, obtundation, or coma and are usually apparent early in the course of illness (within the first hour); consequently, there is little difficulty in recognizing this end of the continuum of heat illness. Signs of organ damage may be more subtle and

develop more slowly; requiring close monitoring of vital signs and laboratory studies. Progressive heat stroke often involves rhabdomyolysis and acute renal failure, occasionally involves liver necrosis and, in advanced stages, DIC. Severe hypotension or sudden cardiovascular collapse occurs in about 5% of cases.

Individuals with heat stroke often have rectal temperatures exceeding 106°F [41°C]. Extreme hyperthermia per se may cause tissue damage at temperatures above 108°F [42°C]. High temperatures also change the kinetics of enzyme and other chemical reactions in the body. However, short-term rectal temperatures of 110°F [43°C] are seen in exertional heat stroke patients with no long-term sequelae if cooling and rehydration occur rapidly, and survival without permanent residua has been reported in at least one patient with a maximum rectal temperature exceeding 115°F [46°C]. In addition, cases of exertional heat stroke also occur at lower rectal temperatures, and many patients with high rectal temperature do not manifest the severe neurologic symptoms or organ damage that define heat stroke.

The temperature elevation in heat stroke is referred to as hyperthermia, not fever. In fever the primary event is an elevation of the thermoregulatory set point. In hyperthermia the regulatory set point remains normal but the thermoregulatory system is overwhelmed and unable to maintain the set-point temperature. When the fundamental cause of hyperthermia is sustained exposure to heat stress with little contribution from exercise, the case is considered *classic heat stroke*. It usually develops gradually, often over a period of days, with slow progression of dehydration and obtundation. Patients often present with marked hyperthermia, hot dry skin, and coma. Classic heat stroke occurs primarily in older people who have diminished cardiovascular reserve, and it has a high mortality. When the fundamental cause of hyperthermia is heavy exercise in hot weather, the resulting illness is considered *exertional heat stroke*. Typically, onset is abrupt, occurring during or shortly after exertion, with orthostatic manifestations (faintness, staggering, and/or visual disturbance) leading to collapse (with or without syncope), followed by confusion, combativeness, delirium, obtundation, and/or coma. This syndrome frequently evolves in minutes. If it is treated immediately by aggressive cooling and rehydration, severe organ damage and mortality are almost always prevented.

Water and electrolyte depletion, acute renal failure, and rhabdomyolysis are common components of heat stroke, and patients must be closely monitored and aggressively treated to minimize morbidity and mortality. Uncommon complications include hepatic necrosis, gastrointestinal bleeding, hypoxemia, pulmonary edema, shock, and DIC. Life-threatening cardiovascular collapse may occur early during the hyperthermic phase, emphasizing the need for immediate cardiac resuscitation capability.

EXERTIONAL RHABDOMYOLYSIS

Exertional rhabdomyolysis is caused by skeletal muscle damage with release of cellular contents into the circulation, including myoglobin, potassium, phosphate, creatine kinase (CK), lactic acid, and uric acid. These usually peak 24 to 48 hours after onset of illness. The severity of rhabdomyolysis can vary from asymptomatic elevation of serum skeletal muscle enzymes to muscle weakness, pain, tenderness, and stiffness with associated myoglobinuria, which can culminate in life-threatening metabolic acidosis, acute renal failure, DIC, and cardiovascular collapse. Although exertional rhabdomyolysis most often occurs as part of exertional heat stroke or heat injury, severe episodes without hyperthermia or encephalopathy are not uncommon.

Severe rhabdomyolysis may present without early muscle pain or tenderness, and muscle numbness may be the only symptom in the first few hours. Most patients develop remarkable muscle tenderness and pain on use, but there is great variability in muscle symptoms despite CK levels as high as 10,000 to 60,000 IU/L. Exercising military trainees frequently develop CK values ranging from 300 to 700 IU/L, but seldom above 1000 IU/L in the absence of heat illness. Other serum tests or enzymes that may be elevated include uric acid, lactate dehydrogenase (LDH), and aspartate transaminase (AST).

If skeletal muscle necrosis is extensive it produces a tissue lysis syndrome with lactic acidosis, hyperkalemia, hyperuricemia, hyperphosphatemia, hypocalcemia, dehydration, acute renal failure, and hypotension. These complications can become life-threatening, especially if DIC produces further organ damage. Fatal complications include acute hyperkalemia, hyperuricemia, hyperphosphatemia, disturbances of calcium metabolism, metabolic acidosis, advanced renal failure, shock, bleeding, myocardial ischemia, and secondary infections. It is extremely

important to closely monitor fluid and electrolyte and acid-base status, because early aggressive parenteral correction of dehydration and electrolyte disturbance is the only effective treatment. The clinical picture may be deceptive, since the patient may manifest minimal clinical symptoms in the presence of profound metabolic abnormalities.

EXERTIONAL ACUTE RENAL FAILURE

The syndrome of isolated acute renal failure with hyperosmolar hyponatremia was often seen when salt tablets were in common use. By restricting oral salt replacement to mealtime consumption of salty foods this syndrome has become quite rare.

EXERTIONAL HYPONATREMIA

In recent years several clusters of cases of hyponatremia ($\text{Na} < 130 \text{ mEq/L}$) have been noted in association with exertional heat illness. These can be severe or fatal, often presenting with nausea and vomiting, followed by seizures and associated brain swelling. This is a form of water intoxication due to excessive water drinking and inappropriate renal retention of water, perhaps due to dehydration and the simultaneous triggering of thirst and vasopressin secretion. This illness occurs primarily in the setting of forced water drinking ($>15 \text{ L/day}$) for prevention of heat illness or to treat early symptoms of heat illness. If rehydration in exertional heat illness requires more than a liter or two of fluid, it should be accompanied by assessment of serum electrolyte levels.

EXERTIONAL HEAT INJURY

Exertional heat injury is a progressive multisystem disorder reflecting collapse of the thermoregulatory system accompanied by organ dysfunction, usually metabolic acidosis, acute renal failure, and and/or muscle or liver necrosis. Patients with exertional heat injury often have milder neurologic symptoms. The absence of delirium, obtundation, or coma distinguishes exertional heat injury from exertional heat stroke. As in heat stroke, organ damage frequently is not manifest at the time of presentation of the patient. During the first hours of illness it may not be possible to distinguish exertional heat injury from heat exhaustion. Therefore, it is essential that all patients with heat illness be thoroughly evaluated for organ dysfunction before release from medical care, with re-evaluation often necessary on the following day.

HEAT EXHAUSTION

Heat exhaustion is a functional multisystem disorder reflecting inability of the circulatory system to meet the demands of thermoregulatory, muscle, and visceral blood flow. It represents, primarily, a syndrome of dehydration without serious metabolic complications. Heat exhaustion is typically thought of as producing minor elevations of rectal temperature (104°F [40°C]), but it can be associated with subnormal or very high rectal temperatures. Increased circulatory demand (the result of increased muscle and thermoregulatory needs) and reduced effective blood volume (the result of thermoregulatory expansion of peripheral intravascular volume and depletion of water and electrolytes through respiration and sweating) result in metabolic acidosis and loss of orthostatic blood pressure control, which often produce exertional (heat) syncope.

Heat exhaustion most often includes both water and salt depletion, and most patients have nearly normal serum sodium levels. Patients need free water replacement, as well as isotonic solutions. In predominant water depletion there are characteristically intense thirst, fatigue, weakness, impaired judgment, hyperventilation with paresthesias, tetany, and hyponatremia. In predominant salt depletion there are characteristically headache, nausea, vomiting, giddiness, generalized muscle cramps, and hyponatremia. However, symptom patterns do not reliably reflect relative deficits of water or salt, and most patients benefit from hydration with normal or half-normal saline.

The symptoms of heat exhaustion can be quite varied (*Table 17-2*) and are rapidly improved by water and salt replacement, a cool environment, and rest. Rapid cooling to a body temperature below 102°F [39°C] is essential. Since heat exhaustion is a functional illness, with no organ or tissue damage, rapid improvement is the rule. Heat exhaustion may progress to heat injury or heat stroke if appropriate measure of rest, rehydration, and cooling are not instituted.

HEAT CRAMPS

Heat cramps are painful migratory skeletal muscle spasms occurring mainly in conditioned persons, at the end of the working day, or in the shower as muscles cool. They are attributed to salt depletion and resultant hyponatremia, and are rapidly reversed with water and salt replacement, usually by oral

solutions containing electrolytes or infusion of normal saline. Prevention is through adequate water replacement and a high salt diet.

TABLE 17-2. Clinical Manifestations of Heat Illness

Nonspecific Symptoms		
Thirst	Hyperventilation	Headache
Weakness	Lack of concentration	Nausea
Fatigue	Impaired judgment	Vomiting
Myalgia	Anxiety	Diarrhea
Cramps	Hysteria	
Progressive Orthostatic Symptoms		
Faintness		Wobbly legs
Dizziness (not vertigo)		Stumbling gait
Visual-blurred vision, tunnel vision, scotomata, blackness		
Collapse (without loss of consciousness)		
Exertional (Heat) Syncope:		
Collapse with brief loss of consciousness		
Severe Symptoms		
Orthostatic or sustained hypotension, shock, cardiovascular collapse, pulmonary edema, acute respiratory distress syndrome		
Metabolic Complications		
Lactic acidosis	Rhabdomyolysis/ myoglobinuria	
Electrolyte imbalances	DIC/bleeding diatheses	Hepatic necrosis
Acute renal failure		
Neurologic Symptoms		
Slow or altered mentation	Agitation, combativeness, lethargy, obtundation,	
Poor concentration	delirium, coma, amnesia,	
Drowsiness, dazed affect	vertigo, seizures, ataxia	
Confusion, disorientation		

DIC, disseminated intravascular coagulation.

MINOR HEAT-ASSOCIATED SYNDROMES

HEAT RASH, PRICKLY HEAT, MILIARIA RUBRA

Heat rash (also known as prickly heat or miliaria rubra) is a pruritic red papular rash, located particularly in areas of restrictive clothing and heavy sweating. It is one of the most common skin problems in hot climates, causing considerable irritation and discomfort. It is caused by inflammation of the sweat glands and blockage of the sweat ducts, perhaps as a result of maceration of the stratum corneum from continuous wetting of the skin by persistent sweating. The rash is an erythematous epidermal vesicular eruption that is pruritic and accompanied by a prickling sensation ("prickly heat") when sweating is provoked. It may interfere with sweating and can therefore be a risk factor for more serious heat illness. Sleeplessness (due to itching) and secondary infection may further aggravate thermoregulation. Treatment is by cooling and drying the affected skin, controlling infection, and managing pruritus, with

resolution of the rash over 7 to 10 days. Rare severe cases with generalized and prolonged rash, miliaria profunda, which has the appearance of goose flesh (Fig. 17-2), may require evacuation to a cooler environment to restore normal sweat gland function.

HEAT EDEMA

Travelers to the tropics may have temporary swelling of the feet and ankles, which is usually mild and disappears within a few days. Edema is due to expansion of blood volume during acclimatization and does not indicate excessive water intake or cardiac, renal, or hepatic disease. Management is to loosen clothing and elevate the legs.

PARADE SYNCOPE

Parade syncope is fainting during prolonged standing, due to inadequate venous blood return to the heart and brain. It can occur in the absence of heat illness, although it may be more common in a hot environment. Syncope occurring during or after work in the heat should be considered evidence of heat exhaustion or heat injury, warranting evaluation of fluid and electrolyte status. Parade syncope is managed by restoring normal blood circulation and minimizing peripheral pooling through a brief period of recumbence in a cool place. Allowing individuals to move about will help prevent parade syncope, and provision of chairs and/or railings will decrease the risk of injury from falling.

Sunburn

Sunburn reduces the thermoregulatory capacity of the skin and should be prevented through adequate sun exposure protection. It should be managed as any other burn, and heat stress should be avoided until the burn has healed.

CLINICAL PRESENTATION AND MANAGEMENT

SYMPTOMS AND SIGNS

Table 17-2 lists, in order of progressive severity, the wide variety of symptoms associated with heat illness. Early in the course of illness, weakness, fatigue, headache, thirst, hyperventilation, impaired judgment, or abdominal or muscle symptoms may predominate. As the illness worsens ortho-

static symptoms develop (e.g., faintness, stumbling gait, blurred vision, narrowed or tunnel vision, or scotomata). Collapse may occur with or without loss of consciousness. Collapse with a brief period of loss of consciousness during or immediately after strenuous exercise is a fainting episode referred to as exertional (or heat) syncope. Syncope must be distinguished from a seizure or coma.

Neurologic symptoms are characteristic in heat illness, particularly in exertional heat stroke. These symptoms are not neurologic deficits relating to specific cranial or peripheral nerves, but represent a generalized encephalopathy affecting mental status. The symptoms range from lethargy and drowsiness through confusion and disorientation to delirium, obtundation, or coma. Amnesia is frequent and there are occasionally seizures and/or persistent ataxia.

CLINICAL MANAGEMENT

Heat stroke is a medical emergency. Early recognition and prompt treatment of heat stroke dramatically reduce the mortality rate. Rectal temperature should be obtained immediately and rapid cooling initiated if above 102°F [39°C]. In addition, there must be close monitoring of vital signs and mental status, aggressive replacement of fluid and electrolytes, and appropriate laboratory workup. Even in milder forms of heat illness immediate measurement of rectal temperature is necessary to help assess severity, the need for rapid cooling, and prognosis.

In controlled settings emergency medical care for heat illness should be arranged in advance through immediate access to medical support, including capability for measurement of rectal temperature, clinical assessment, and ice water cooling. In settings where heat illness is common, strenuous exercise or work should not be conducted without medical capability present on-site. Delay in treatment may result in much more severe illness, with resultant complications, organ damage, and increased morbidity and mortality.

COOLING

Body cooling should be accomplished as quickly as possible, using whatever means are available under the circumstances. The clothing should be loosened or removed, and evaporative cooling maximized. Methods for cooling include immersion in cold water, showering, and soaking the clothing, as well as using ice bags or a cooling blanket. The cooler the water the more rapidly the cooling process progresses, and ice bags placed over major arteries

may enhance cooling. Briskly massaging the body helps prevent cutaneous vasoconstriction, which may impede core heat dissipation. Fanning can be accomplished manually, using electric fans, or by placing the patient under rotating helicopter blades (a military field expedient that appears to be very effective). There has been controversy concerning whether use of tepid water with fans or use of ice water is clinically appropriate. Clinical experience suggests that ice water may cause coronary spasm in people with coronary atherosclerosis but is not harmful in those without such disease. Ice water has been demonstrated to lower rectal temperature about twice as rapidly as use of tepid water with fans. We therefore recommend that ice water be used first on patients under 35 years of age and that tepid water be used first on older patients, especially those with a history of angina or coronary heart disease.

Rapid cooling should continue until the rectal temperature remains below 102°F [39°C]. After reaching this temperature, cooling can proceed more slowly until the rectal temperature remains below 100°F [38°C]. Regardless of the method of cooling, the degree of organ damage and metabolic and neurologic complications is directly related to the magnitude and duration of elevated body temperature--patients who cool slowly tend to have more serious illness.

REHYDRATION

Rapid parenteral administration of 1 to 2 L of normal saline has been shown to enhance cooling. Rehydration should include restoration of water and electrolytes and re-establishment of normal acid-base balance. Generally 1 L of isotonic fluid is given immediately, with an additional 2 or 3 L as required. Some free water replacement is necessary, as well as normal saline solutions. A clinical decision to give more than 2 L of IV fluids constitutes an indication for laboratory determination of serum electrolytes. Management plans should include prompt laboratory evaluation, setting a desired level of fluid intake, maintaining records of fluid intake and output, and frequently monitoring vital signs to confirm that postural blood pressure control is returning to normal.

LABORATORY EVALUATION

In the circumstance of exercise-induced mental dysfunction, prompt laboratory evaluation is necessary to identify significant metabolic problems that may initially be asymptomatic: (1) exertional rhabdomyolysis, (2) acute renal failure, (3) depletion of electrolytes and water, (4) exertional hypo-

TABLE 17-3.
Recommended Laboratory Workup for Heat Illness

Blood Count (CBC)	Hemoglobin, hematocrit, white blood cell count, platelet count
Urinalysis	Specific gravity, pH, evidence of myoglobin
Serum Chemistries (Heat Panel)	Sodium, potassium, chloride, bicarbonate, glucose, blood urea nitrogen, creatinine, uric acid, creatine kinase, aspartate transaminase, lactate dehydrogenase, alanine aminotransferase
If Severe	Arterial blood gases; calcium, phosphate; prothrombin time, partial thromboplastin time, fibrin split products, fibrinogen

Table 17-4. Classification and Stratification of Heat Illness

Category	Dehydration	CNS	Renal	Lysis
1	Nonspecific symptoms	Normal	Cr: \leq 1.4 mg/dL	CK: $<$ 700 IU/L
2	Orthostatic symptoms	Lethargic, slow mentation	Cr: 1.5-1.7 mg/dL	CK: 700-1200 IU/L
3	Exertional syncope	Amnesia, confusion	Cr: 1.8-1.9 mg/dL	CK: 1200-4,000 IU/L
4	Orthostatic hypotension	Delirious, ataxia/vertigo	Cr: \geq 2.0 mg/dL	CK: 4-10,000 IU/L Progressive muscle symptoms
5	Shock	Seizure, obtunded, coma	Acute renal failure	CK: \geq 10,000 IU/L Metabolic changes

Clinical Management

- Category 1: Follow-up is not usually needed.
- Category 2: Clinic and / or laboratory follow-up may be indicated.
- Category 3: Clinic and laboratory follow-up is probably indicated
- Category 4: Hospitalization may be indicated.
- Category 5: Hospitalization is indicated.

CNS, central nervous system; CK, serum creatine kinase; Cr, serum creatinine.

glycemia, and (5) metabolic acidosis. Close monitoring of vital signs and serum chemistries is essential, since clinical symptoms may not reflect profound metabolic abnormalities. The recommended laboratory workup includes a complete blood count, urinalysis, and serum chemistries (Table 17-3). These assess dehydration, electrolytes, acid-base balance, renal function, and muscle and liver cell lysis. Maximum laboratory abnormalities often do not appear until 24 to 48 hours after the onset of illness, necessitating follow-up laboratory assessment the next day, except in mild cases.

The standard measures for handling an unconscious or confused patient should be taken. Severely ill patients will require invasive monitoring and management in an intensive care unit. Classification in terms of severity in each major organ system is more useful than classification by specific syndrome and helps to guide clinical management (Table 17-4). This scheme applies to otherwise healthy young adults, and may require adjustment for elderly or very young patients.

Many patients appear to have mental and perhaps physical impairment for several days following even a moderate episode of heat illness. This requires activity restriction and continued follow-up. Patients having only moderately severe heat illness have been reported to have poor judgment and difficulty concentrating lasting at least 3 days following the episode, despite appearing completely well.

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FOOT BLISTER CARE AT NIJMEGEN 2000

David L. Hamilton, PA-C

ABSTRACT

Forced foot marches have been a staple of soldiering throughout history. Unfortunately, many soldiers' and leaders' desire to cover long distances quickly can cause friction blisters to form on soldier's feet. Many techniques have been used to toughen feet, treat blisters and enable soldier to "Charlie Mike" (continue the mission) with varying results. This work presents an alternative blister care procedure to traditional moleskin donuts and pressure relief techniques.¹ These treatments may cause blisters to enlarge with continued marching, and may require frequent halts for adjustment / reapplication. By treating soldiers with severe early blister formation by taping, and pre-taping feet prone to blistering prior to long marches, soldiers may be returned to marching fitness during a time of need.

(Translation: Keeps you from having to carry them out!!!)

INTRODUCTION

While participating in the *Nijmegen 4 Days' March* in July 2000, the author observed a unique foot blister treatment system used by the Dutch Red Cross to provide medical support to thousands of marchers. This foot taping and blister management system limits further blister formation and enables marchers to continue marching for several consecutive days; marching 26 miles daily in combat uniform and equipment. The tape is sturdy enough to leave on for several days, if need be, to facilitate continuous operations, making it an ideal system for special operating forces on long foot movements.

PURPOSE

This work presents an alternative blister care procedure to traditional moleskin donuts and pressure relief techniques^{1,2}. These treatments may cause blisters to enlarge with continued marching, and may require frequent halts for adjustment / reapplication. This work presents an alternative blister care procedure to traditional moleskin donuts and pressure relief techniques¹. These treatments may cause blisters to enlarge with continued marching, and may require frequent halts for adjustment / reapplication. By treating soldiers with severe early blister formation by taping, and pre-taping feet prone to blistering prior to long marches, soldiers may be returned to marching fitness during a time of need. (Translation: Keeps you from having to carry them out!!!)

BACKGROUND

The *Nijmegen 4 Days Marches* began in 1909, with marchers walking 140 kilometers in 4 days. Military groups now march 160km in 4 days. The walks have been held annually since 1909, except during the war years of 1913-14 and 1940-45. In the 2000 march, 41,000 people from 51 countries started and 36,377 finished.³

During the *4 Day's March*, military contingents billet at Camp Heumensoord. The Dutch, German and British Militaries establish formal camps and medical treatment facilities, the largest of which is the Dutch Field Treatment Station, roughly the size of a US Forward Support Medical Company Clearing Station, with the addition of Physical therapy.

American soldiers have no formal support (it was withdrawn after 1996 for various personnel draw-down and deployment reasons), and fall in with the Small Contingents formations. Many US units and individual soldiers still participate with their own funding (in 2000, 504 US military personnel started and 470 finished the march).⁴

PROCEDURE

US soldiers receive their medical care from unit medics and the Dutch treatment tent, but as there may be 300-400 soldiers with blisters evaluated each evening at the treatment tent, waiting times are long (up to 3 or 4 hours) During this waiting time, soldiers are signed in and issued a number, and given an

approximate waiting time, so they may return at that time.

The Dutch "blaren" (blister) treatment area contains 20 to 30 treatment tables, staffed with Dutch medical specialists and Dutch Red Cross personnel. Several doctors are available for the specialists to consult regarding serious erosions and infections. Each area is well stocked with these supplies:

- Finger lancets
- Tincture of benzoin
- Leukoplast ¼ " tape
- Large cotton tip applicators
- Mild betadine solution foot wash
- Cotton balls
- 2x2 gauze
- 4x4 gauze
- Talc or foot powder
- Duo-Derm
- Elastic Bandages
- Epsom Salt or Domboro foot soaks
- Sterile Normal Saline Irrigation
- Foot basins
- Ice (if available)

The system takes approximately 30 to 40 minutes to tape a pair of blistered feet.

The evaluation of the blistered foot requires the determination if the marcher can medically continue marching. Contraindications to continued marching include deep cellulitis surrounding vesicles; foul smelling purulent bullous fluid, deep dermal erosion and repeated bleeding from vesicle after initial debridement. For this article we will classify march blisters in 4 types: (*See Figure 1*)

- sealed vesicles filled with bullous fluid
- sealed vesicles with purulent fluid
- ruptured vesicles with intact roof or de-roofed vesicles
- deep dermal / sub dermal abrasions.

Soldiers are initially evaluated with a brief history, including location and severity of pain, ability to walk, types of socks and boots used, previous treatments, allergies, and significant past medical history. If serious injuries are discovered in the history, a medical officer is summoned to further evaluate the patient. If no significant medical problems are discovered, the medical specialist proceeds to examine the foot and ankle, with particular attention to the skin of toes and inter-digital spaces, heel, and ball of foot. Cleansing of the foot with a mild disinfectant solution is followed by removal of old tape, moleskin and tincture with tape remover or acetone. The skin is then re-inspected for signs of erythema,

severe edema or lymphangitis, indicators of cellulitis. If cellulitis is detected, the soldier is usually removed from marching status and treated with appropriate bed rest, limb elevation and antibiotics.

INITIAL TREATMENT OF VESICLES PRIOR TO TAPING (*See Figure 2*)

INTACT (SEALED) VESICLES:

After examining and cleansing to foot, the top of the intact vesicle is disinfected with an alcohol swab. The selection of the drainage site is important, as drainage needs to occur during sleep as well as during marching. Usually a puncture on the posterior aspect of the vesicle will allow for appropriate drainage.

The medical specialist uses a standard finger lancet to puncture the vesicle wall close to the base. The lancet prevents too deep of a puncture, just through the vesical roof. A very small puncture is made, and the vesicular fluid is expressed using the stick of the "Texas Q-tip" (large cotton tipped applicator). This process is extremely painful for the patient. The firm rolling of the plastic handle of the applicator irritates the extremely sensitive vesicular base, even though the roof is still intact. This rolling must be done repeatedly to ensure all of the vesicular fluid is removed.

If purulent fluid is expressed from the vesicle, the vesicular roof must be removed to allow debridement of the wound and further evaluation of the depth of the infection. If, after deroofting the vesical, there is no deep-seated infection and the surrounding tissue is not erythematous, tender and no lymphangitis is seen, the vesical may be treated using the procedure below.

DEROOFTED OR TORN VESICLES:

If a vesical roof has torn, or is partially missing, the area is cleansed as above. The loose epidermis must be removed, as it no longer serves a purpose as a physiologic bandage. Use sterile scissors (iris, curved mayos or one point sharps) to fully trim the loose skin, beveling the edge of the erosion to prevent further friction injury. After debridement of the vesicle roof, cleanse the blister base with normal saline irrigation. This area is very sensitive, but gentle irrigation is vital to prevent infection. If active bleeding, extreme beefy redness or purulence exists at the blister base, then the soldier should be treated for cellulitis as outlined above.

DuoDerm is a soft polymer that becomes more flexible with body heat. It is used by the Dutch

Red Cross to provide an airtight dressing over the sensitive blister base, and to fill the empty space left by the vesicle's debrided epidermis. Cut a section of 1/8" thick DuoDerm the same size as the eroded cavity, and warm it (or have the patient warm it) in a gloved hand for 5 minutes. When the film is soft, peel the backing and apply to the blister base. This provides a sterile dressing that soothes the wound. Ensure there is no "double thickness of DuoDerm and skin which may cause additional friction pressure after dressing and taping.

DEEP DERMAL ABRASIONS

Deep dermal friction abrasions occur when the soldier's level of "Drive-On" exceeds the anatomical structural resilience of the skin and dermis. Deep dermal friction abrasions begin as simple "hot spots" of increased friction, progress to simple friction blisters, and on to deep dermal erosions. These can usually be recognized active bleeding, deeply abraded tissue, and extreme tenderness. Soldiers may recognize there is a problem when they notice bloody socks or blood in their boots.

It is very difficult to restore deep dermal erosions to marching status. It is more appropriate to remove the soldier from weight bearing on the affected foot for several days, dress with a bulky dressing and topical antibiotic, twice daily Domboro's soaks, foot elevation and observe wound for signs of cellulitis.

DUTCH RED CROSS BLISTER TREATMENT TAPING TECHNIQUE (See Figures 3&4)

For forefoot vesicles, Place the patient in a supine position with the feet extending slightly over the table edge. Tape the entire forefoot, beginning at the base of the toes and continuing to the mid arch. For heel vesicles, place the patient in the prone with the feet extending over the edge. Start at the top of the Achilles' fossa and continue to the mid arch.

Leukoplast 1/4" dressing tape is used because of its thinness and flexibility to molding to the foot. Other thin tapes may be used, but ensure it is cloth and non-stretch.

Begin by measuring the Leukoplast by pulling from the roll and placing on the area to be taped by the non-adhesive side, then cut to the appropriate length (enough to go from the start of the plantar-textured skin on the upper medial foot, to where that type of skin ends on the opposite side of the foot.) It is important to use tape that is long enough; so cut the tape longer rather than shorter (it can be trimmed if it is too long) on the medial and dorsal side of the foot.

You will need different lengths of tape as you tape the different areas of the foot, so measure each area of the foot treated to ensure the proper length of precut tape.

When applying tape, stick the middle of the tape to the foot first, then carefully smooth it to the skin to ensure no wrinkles are applied. Start your next strip by overlapping the previous strip by 1/3 the width of the tape. Continue taping until the entire area is covered, and then ensure no wrinkles or excessive layers of tape are present (these may cause new friction points or hot spots). Tape carefully around blistered toes, cutting wedges from the tape as needed to prevent friction points.

End state must be a smooth, tight, even taping of the affected section of the foot. When this is achieved, dust the taped area with talc to prevent the tape edges from sticking to the socks. Ensure the borders of the taped area are well adhered to the skin and add additional tincture to those areas as needed to ensure the tape is secure.

Advise the soldier to dust the tape with talc every time a sock is changed, and to be aware of any fluid build up in the old blister pockets. These may be drained without removing the tape, a small area of tape may be peeled back from the drainage site, the skin disinfected, a sterile lancet or 18 gauge needle used to drain the vesicle, and reapply the old tape over the site (or, if the tape is non-adherent, apply a section of new tape, ensuring no wrinkles).

The soldier should be instructed to return to medical care in case of extreme pain, purulent or bloody exudate from the blister, or signs of infection occur.

EXAMPLES OF BLISTERS TREATABLE BY TAPING

Figure 1⁵

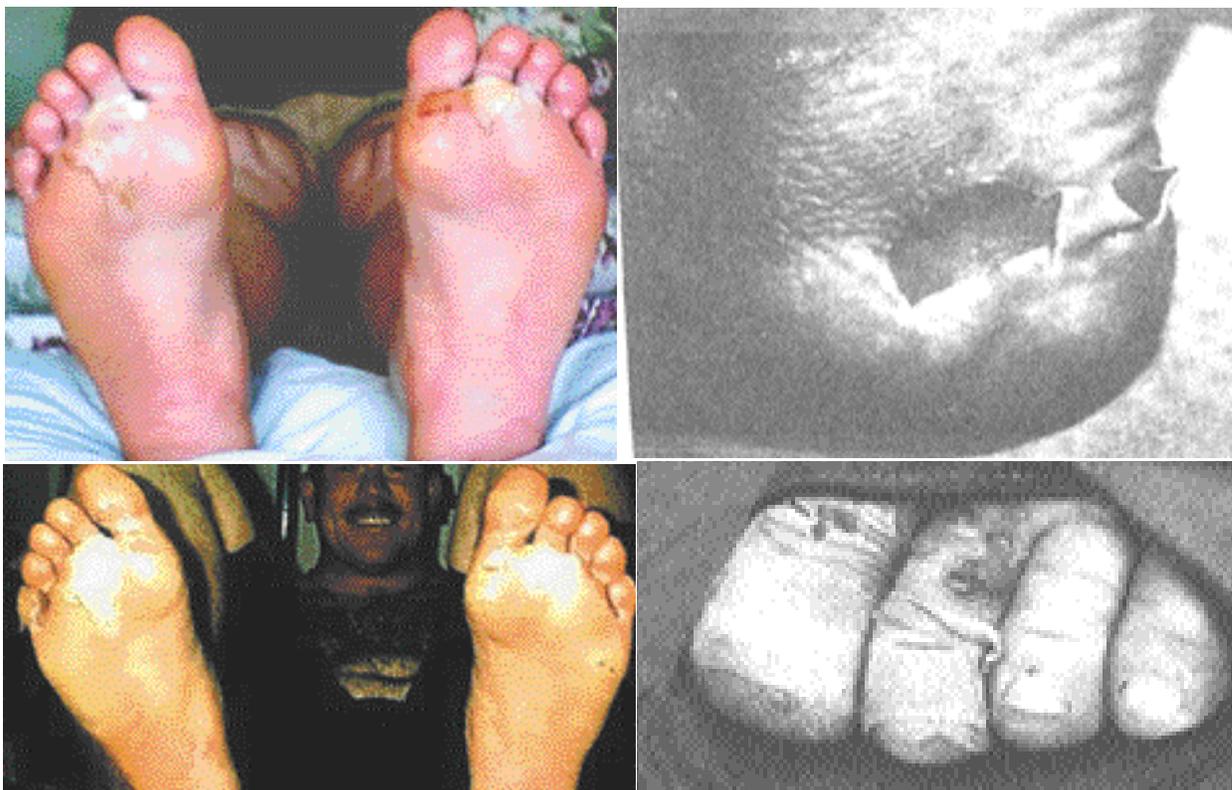
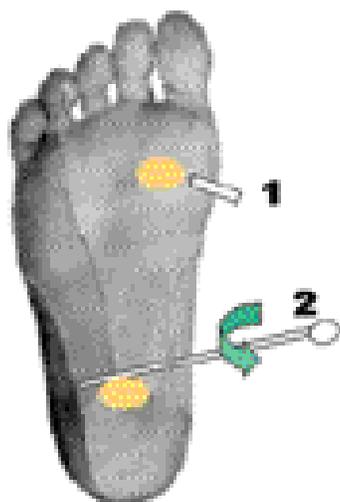


Figure 2

PREPARATION FOR TAPING



- **Cleanse foot with mild disinfectant wash**
- **Remove old tape and moleskin**
- **Sterilely drain vesicle with lancet**
- **Roll vesicular fluid out completely with "Texas Q-Tip"**
- **Dress Denuded Areas with DuoDerm.**
- **Wipe area to be taped with tincture of benzoin**
- **Begin taping**

Figure 3

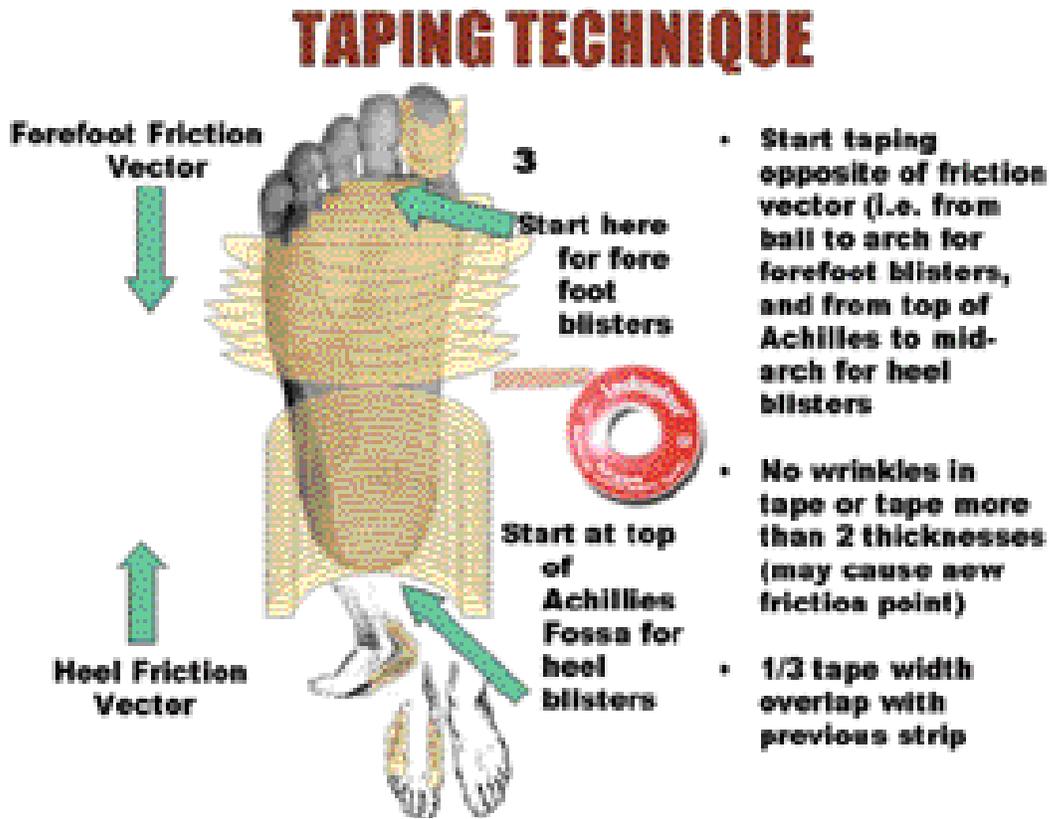
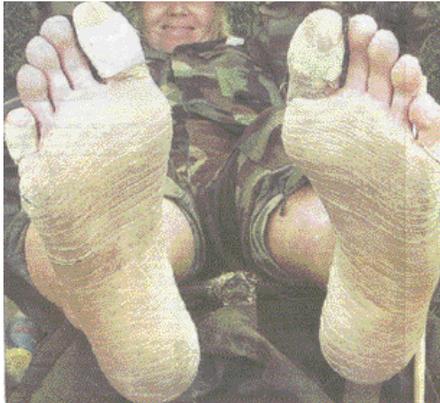


Figure 4
FINISHED TAPING



Conclusion

Proper training, boot fitting, sock and insole selection and stopping to fix "hot spots" when they occur are the essential for the PREVENTION of blister formation during long military foot marching. When blisters do occur, a focused evaluation will determine the severity of the injury and the fitness of the

soldier for continued marching. If the blister and surrounding tissue is not severely infected, the Dutch Red Cross Foot Taping Technique will allow the motivated soldier to "Continue to March" without additional serious injury. This epitomizes the Nijmegen 4 Day March motto of Willen is Kunnen (Willing is Can!)

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Going Beyond the “Thin Air”

An Understanding of Physiologic Acclimatization and The Pathogenesis of High-Altitude Related Injuries

First in a Two Part Series

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ABSTRACT

This is the first of a two part series that discusses High Altitude acclimatization and pathogenesis. Military operations at high altitudes have existed for centuries and are currently being conducted now in parts of the world. This paper reviews a brief history, physiological changes leading to acclimatization, types of injuries and the use of adjunctive treatment modalities to better conserve the human component combat weapons system.

OBJECTIVES (FOR PARTS 1 AND 2)

1. Explain the compensatory mechanisms that take place in the physiologic adaptation and acclimatization process as it relates to high altitude.
2. Be able to recognize the signs and symptoms of high altitude illness and render the appropriate treatment modalities.
3. Discuss indications and contraindications along with side effects of the Gamow Bag.

Complete test on page 54--Answer sheet on page 57

Completion of this article and test offers 1.25 CME and 1.4 CNE/CEH

DISCLOSURE:The presenters have indicated that, within the past two years, they have had no significant financial relationship with a commercial entity whose products/services are related to the subject matter of the topic they will be addressing or a commercial supporter of this educational activity.

WE DEDICATE THIS ARTICLE TO THE MEN OF THE 5TH SPECIAL FORCES GROUP (AIRBORNE) FOR THEIR GALLANT BRAVERY, DEDICATION TO DUTY, AND CONSUMMATE PROFESSIONALISM IN SUPPORT OF OPERATION ENDURING FREEDOM IN AFGHANISTAN. THE GROUP EPITOMIZES THE QUIET PROFESSIONAL WHOSE CREED IS "TO FIGHT SO OTHERS MAY REMAIN FREE."

DE OPPRESSO LIBER

INTRODUCTION

The survival of a species primarily depends on its ability to adapt to acute and chronic stress. Darwin elaborated on this concept when he described his principle of the survival of the fittest in *The Origin of the Species by Means of Natural Selection*. The human body requires a constant supply of oxy-

gen in order to survive. Decreased availability of oxygen in the ambient air (hypobaric hypoxia) is one of the environmental stresses unique to high terrestrial altitude. High altitude presents a natural stress where a concomitant decrease in atmospheric barometric pressure correlates directly with increasing altitude. Consequently, a decrease in the partial

pressure of oxygen is present at each step of its transport from the inspired ambient air to the tissues where oxidative metabolism supports life. The partial pressure of this vital gas diminishes as the oxygen molecules move from air to blood to tissues. The decreased availability of oxygen causes a reduced partial pressure lowering the oxygen supply to the body tissues, which causes altitude illness and the decline in mental and physical performance. Impairment of this process can arise from both environmental and pathologic causes, further contributing to the hypobaric hypoxic state.

Unique to this information article is a collaborative overview explaining the effects of hypoxic stress placed on the human body, which creates an acid-base metabolic and respiratory imbalance. If left uncorrected, this imbalance, coupled with prolonged hypoxemia, causes activation of the inflammatory cascade producing eicosanoids through arachidonic acid synthesis. It is this inflammatory cascade which helps contribute to the edema seen in the brain and the lungs. Given its ubiquitous effects, a basic understanding of hypobaric hypoxia is essential for medical personnel who support military units operating in high mountainous regions.

The purpose of this article is to:

- Review the compensatory mechanisms that take place in the acclimatization process responsible for the transport of oxygen to the tissues when immersed in a hypobaric hypoxic environment;
-

HISTORICAL BACKGROUND

For most of its approximate 4.6 billion years, the earth has maintained a very low-concentration oxygen environment. About a half billion years ago, photosynthetic activity increased, and about 100 million years ago the oxygen level was sufficient to permit life of multi-cellular organisms.¹ Subsequently, the fraction of oxygen in the atmosphere up to 70 miles above the earth's surface is thought to have reached a plateau at a fraction 0.2093.² The partial pressure of oxygen in the inspired ambient air, therefore, is simply 0.2093 x the barometric pressure. Although it is possible for climatic conditions above 16,000 ft to sustain life, prolonged survival at this altitude is not likely. However, it is possible for humans to thrive at altitudes greater than 8,000 ft. Nearly thirty million people live at these altitudes primarily in the Rocky Mountains of North America, the Andes of South America, the Ethiopian Highlands of East Africa, and the Himalayas of

South-central Asia.³

Speculation about the effects of altitude and the lack of oxygen in the air can be traced back to the late sixteenth century. Jose de Acosta, a Jesuit priest, who in 1590 accompanied the Spanish conquistadors during their exploration of Peru, first described the ailments of extreme altitude. He hypothesized that it was the "thin air" that was responsible for headache, dyspnea, and light-headedness that often occur upon acute ascent. John Mayow, a pioneer in the discovery of oxygen, conducted his well known sealed bell jar and mouse experiments in 1674. When he burned a candle in a jar with mice in it, he found the mice died when the candle burned out. One hundred years later, Lavoisier in France and Priestley in England were able to generate oxygen, which they determined makes up about 21% of the atmosphere.⁴

High-altitude physiology burgeoned in the latter half of the nineteenth century. A French physiologist, Paul Bert, is credited with being the father of high-altitude research. His steel bell chambers allowed ascent by decompression. A prodigious number of field studies were conducted on Pike's Peak and in the Andes. Landmark reviews by Schneider, Barcroft, and Haldane give an excellent perspective on the extraordinary work that was being done early in the 20th century.⁴ This article builds on the last 30 years of research that evolved from the important work of such pioneers.

THE EFFECTS OF BAROMETRIC PRESSURE AND THE AVAILABILITY OF OXYGEN

There is a curvilinear reduction in the ambient barometric pressure with increasing altitude. The exact magnitude of the reduction is determined by the gravitational distribution law. It states that there is a difference in barometric pressure for a given level of elevation, which is influenced by the combination of elevation, latitude, season, and weather. A concomitant reduction in the partial pressure of oxygen becomes physiologically significant due to the decreased barometric pressure in the ambient air. The progressive decrease in the partial pressure of oxygen means there is less actual oxygen available for respiration. The primary factor limiting the amount of oxygen supplied to the body tissues through respiration is the reduced partial pressure of atmospheric oxygen at high altitude. The percentage of oxygen does not change. It remains a constant, which is approximately 21%.

It is the diminution of barometric pressure associated with increasing altitude that causes a reduction in alveolar oxygen and, consequently, a decrease in arterial oxygen content.

The U.S. Army Research Institute of Environmental Medicine explains the relationship of decreased ambient oxygen to altitude illness and performance decrements.

The relationship of decreased ambient oxygen to altitude illness and performance decrements provides a classification of altitude exposure based upon arterial oxygen content and its physiologic effects. Sea level to 5,000 ft is considered low altitude. Arterial hemoglobin saturations are generally above 96% in healthy people at these altitudes. Moderate altitude extends from 5,000 to 8,000 ft where arterial hemoglobin saturation is normally above 92% and any effects of altitude are mild and temporary. High altitude extends from 8,000 to 14,000 ft. At these elevations arterial oxygen saturation is in the "knee" of the oxygen-hemoglobin saturation curve, ranging from approximately 92% down to about 80% saturation. Altitude illness and performance decrements are increasingly common in this range. Altitudes above 14,000 ft produce hemoglobin saturations that are on the "steep" portion of the oxygen-hemoglobin saturation curve where a small decrease in oxygen tension results in a relatively large drop in hemoglobin saturation. The region from 14,000 to 18,000 ft is classified as very high altitude. Altitude illness and performance decrements are the rule at these altitudes. Regions above 18,000 ft to 29,028 ft are classified as extreme altitude.⁵

HUMAN PHYSIOLOGIC ACCLIMATIZATION TO A HYPOBARIC HYPOXIC ENVIRONMENT

Individuals who ascend from lower altitudes trigger a cascade of integrated physiologic responses when placed in a sustained hypobaric hypoxic environment at elevations over 6,000 ft. These changes function to increase oxygen supply to body tissues. The body systems that are most noticeably affected are those related to oxygen delivery, i.e., the cardiovascular and respiratory system. A number of different processes are involved in the body's oxygen delivery system: ventilation, matching of ventilation with adequate pulmonary perfusion, diffusion of oxygen from the gas to the blood phase, proper circulation, tissue diffusion, and oxidative metabolism in the cell. Many compensatory mechanisms improve oxygen delivery when its concentration is

reduced in the air that we breathe at altitude.

Acclimatization occurs as a two-part process. An immediate response involves the heart, lungs, blood vessels, and kidneys. Secondly, there is a delayed response involving the hematologic system and the tissues. This is characterized by increased erythropoiesis and increased oxygen delivery to the tissues. Coupled with this increase in hematocrit are altitude-induced changes at the tissue level including an increase in capillary density and the number of mitochondria in the muscle. Over time, the series of changes produce a state of physiologic adaptation termed acclimatization. Altitude acclimatization primarily involves submaximal aerobic performance. For example, if a person is capable of running a six-minute mile (max VO₂) at sea level, that individual will not be able to reproduce that same effort at the summit of Pike's Peak at 14,100 ft. However, if that same individual can jog an 8-minute mile at sea level, that person will eventually be able to jog an 8-minute mile at the summit of Pike's Peak after about one month of proper acclimatization. Maher and associates, after conducting studies on eight well-conditioned athletes at exposure to 14,000 ft, suggested endurance exercise capacity improved over time due to improved tissue oxygenation and aerobic capacity as reflected by a diminution of anaerobic by-products, i.e., blood lactate concentrations.⁶ The body's ability to maintain an aerobic state at altitude under conditions of hypoxic stress enhances performance due to the greater yield of ATP produced via the glycolytic pathway. There are 38 moles of ATP generated via aerobic metabolism as compared to only two moles of ATP generated via anaerobic metabolism. The time course and success of acclimatization is a function of the interaction between the unique physiologic characteristics of the individual and the magnitude of hypoxic stress as defined by the elevations gained in the speed of ascent. Once acquired, acclimatization is maintained as long as the individual remains at altitude, but is lost upon that person's return to lower elevations within three to seven days.

The sequence of physiologic changes that produces acclimatization to high altitude takes time to complete. For most individuals at high to very high altitudes, 70% to 80% of the respiratory component of acclimatization occurs in a week to 10 days. Approximately 80% to 90% of overall acclimatization is accomplished by three weeks to a month.⁵ It should be noted, however, that maximum

acclimatization might take months to years. There does not seem to be any way to accelerate this process. Some individuals acclimatize more rapidly than others; whereas, others appear not to acclimatize at all. Unfortunately, there is no reliable way to identify these individuals except by their experience during previous altitude exposures. As previously noted, the amount of time required for a person to become acclimatized is a function of that individual's physiology and the magnitude of the hypoxic challenge as defined by the rate of ascent and the altitude attained.

RESPIRATORY PHYSIOLOGY

The ventilatory response to high altitude is the most immediate and notable physiologic response to occur upon ascent. The oxygen and carbon dioxide concentrations in alveolar air are a direct reflection of barometric pressure and alveolar ventilation. Rahn and Otis in 1946 clearly demonstrated that as barometric pressure decreases, ventilation increases to minimize a drop in alveolar oxygen pressure.⁷ This was evidenced by an increase in tidal volume and respiratory rate, increasing respiratory minute ventilation. The increase in ventilation is achieved initially more by an increase in tidal volume than an increase in frequency, but over time, frequency also increases.⁸ This hyperventilation that ensues causes a respiratory hypocapnic alkalosis but also is an attempt to increase alveolar oxygen partial pressure. The first consequence of the reduction in alveolar oxygen caused by the reduced partial pressure of oxygen at high altitudes stimulates peripheral chemoreceptors found adjacent to the aortic arch and in the carotid body at the bifurcation of the common carotid artery. Carotid body output is inversely proportional to the decreased partial pressure of oxygen in the blood, which leads to hypertrophy of the glomus cells of the carotid body.^{9,10} Hornbein demonstrated that neural discharge from the carotid body had a hyperbolic relationship with decreasing partial pressure of oxygen in arterial blood; this relationship is similar to the ventilatory response to hypoxia.¹¹

On ascent the lung undergoes a number of changes that affect pulmonary mechanics. The changes include a decreased vital capacity, secondary to increased blood flow and central blood volume, an increase in residual volume, and decrease in lung compliance.^{12,13} These changes are ephemeral and usually resolve to leave the sojourner with normal lung function.

Upon ascent to high altitude, increased ventilation results in an increased alveolar oxygen partial pressure. The partial pressure of oxygen in the arterial blood (PO₂) depends on an optimal matching of ventilation and perfusion (VA/Q) in the lung and diffusion of oxygen to the red blood cell. The initial increase in ventilation during acute exposure is matched by an increase in cardiac output and pulmonary perfusion.¹⁴ Hypoxic pulmonary vasoconstriction at the arteriolar level on ascent to high altitudes results in better perfusion to previously underperfused areas. Chronic constriction results in smooth muscle hypertrophy and pulmonary hypertension.¹⁵ A number of mediators such as histamine, prostaglandin, bradykinin, serotonin, angiotensin II, and the transport of calcium at the smooth muscle membranes may be responsible for the pulmonary vascular constriction.¹⁶ Schoene and Hornbein summarize the work of Rotta, Fishman, Dawson and Grover to explain the effects of ventilation-perfusion matching at altitude.

Hypoxia causes pulmonary artery constriction and pulmonary hypertension. This response functions advantageously at rest by improving VA/Q matching and gas exchange. The increased pulmonary vascular resistance causes redistribution of blood flow such that areas of the lung that are usually poorly perfused at sea level, for example, the apices, gain perfusion under hypoxic conditions, resulting in greater homogeneity of VA/Q. Using radio-nuclide scanning, Dawson and Grover found that healthy, long-term residents at an altitude of 3100 meters had a more uniform VA/Q distribution than residents at sea level.⁴

At extremely high altitude, diffusion of oxygen from the alveolus to the blood appears to be the rate-limiting step of oxygen transport. Oxygen flux is dependent upon four variables: the pressure gradient for oxygen from the alveolus to the pulmonary capillary blood, the diffusion capacity of alveolar capillary membrane, capillary blood volume, and the surface area for gas exchange. Several factors may result in an increased alveolar-arterial PO₂ difference at extreme altitude accentuated by extreme exercise impairing oxygen diffusion at the alveolar-capillary level. First, as one ascends, the decreasing partial pressure of oxygen due to the decreased ambient barometric pressure results in a decreasing alveolar to pulmonary capillary PO₂ gradient. Second, at altitude, even the resting pulmonary transit time of 0.75

seconds may not be adequate for full equilibration of oxygen diffusion to take place at the alveolar-capillary junction.

CARDIOVASCULAR PHYSIOLOGY

The next aspect of oxygen delivery affected by sustained hypobaric hypoxia is the transport of oxygen from the lungs to the tissues. Changes occur in both the oxygen carrying capacity and distribution of cardiac output. Upon acute exposure to hypoxia, an increase in cardiac output helps sustain oxygen delivery in spite of a decreased arterial oxygen content. The increase in cardiac output (C.O. = heart rate [HR] x stroke volume [SV]) is the result of an increased heart rate with little change in stroke volume.¹⁷ The initial increase in cardiac output is driven by a hypoxia-stimulation of the sympathetic nervous system activity affecting the sino-atrial node of the heart. Sympathetic activity also causes increased systemic blood pressure, peripheral vasoconstriction, and an increased basal metabolic rate. The slight decrease in stroke volume is due to the loss in plasma volume caused by a movement of fluid out of the vascular compartment into the interstitial and intracellular compartments, diminishing preload to the right atrium of the heart. Over the next few days at high altitude, cardiac output then decreases as a result of this decrease in stroke volume caused by a 10% to 20% loss of plasma volume.⁵ The result of the loss of plasma volume is a relative increase in hemoglobin concentration and an improved oxygen carrying capacity of the blood. With sustained altitude exposure, sympathetic activity decreases and plasma volume tends to recover, which causes a decrease in cardiac output over time. As acclimatization proceeds over the next few weeks, hemoglobin concentration and blood volume increase due to erythropoietin stimulation from the kidney, and oxygen transport is improved by an increase in stroke volume and arterial oxygen content.

At submaximal workloads, the relationship of cardiac output to work rate remains similar to that at sea level. With acclimatization, maximal cardiac output is decreased at high altitude due to a decrease in maximal heart rate and stroke volume.¹⁸ The lower maximum heart rate in sojourners is speculated to be due to a hypervagal tone, direct hypoxic myocardial depression, or an impairment of nodal or bundle branch conduction.

RENAL PHYSIOLOGY

Upon ascent to high altitude, a natural diuresis occurs due to an increased glomerular filtration rate that occurs from the increased cardiac output. Within several days at altitude the kidneys compensate for the respiratory hypocapnic alkalosis by increasing excretion of bicarbonate in the urine. The purpose of this is to help correct the acid base imbalance and restore the pH of the blood closer to neutrality (7.35-7.45). The final effect the kidney has on improved acclimatization is one of hormonal influence. The kidney secretes a hormone called erythropoietin, which causes the bone marrow to increase red blood cell production. Although erythropoietin stimulation occurs within hours from the start of sustained altitude exposure, the increase in red cell production it causes is not measurable for up to three to four weeks.⁵

HEMATOLOGIC ACCLIMATIZATION

The pulmonary and systemic vasculature is the conduit for transport of oxygen from lung to tissue, while the red blood cell and its hemoglobin molecule are the vehicles on which the oxygen rides. Both hemoglobin and the red blood cell undergo changes that improve oxygen transport to the tissues. The two basic adaptations are: 1) an increase in the number of red blood cells (polycythemia), which increases the oxygen carrying capacity of the blood, and 2) an altered affinity of hemoglobin for the oxygen molecule.⁴ Hemoglobin concentration increases within a day or two of ascent and continues to rise to a plateau in two or three weeks. This initial rise is due to hemoconcentration resulting from the diuresis that occurs upon acute ascent. The continued increase results from bone marrow erythropoiesis secondary to hormonal stimulation from the kidney. Hypoxia by itself is the primary stimulus for this hormonal release (erythropoietin) by the kidney. The erythropoietic response to a given level of hypoxia is extremely variable among individuals.¹⁹

Serum erythropoietin levels increase rapidly within the first day of ascent to high altitude, and then start to decline within a few days as acclimatization progresses.²⁰ Both red blood cell volume and total blood cell volume increase with a decrease in plasma volume at high altitude.²¹ The changes increase oxygen content and oxygen delivery in the blood. The erythropoietic response quickly ceases

upon descent and within three weeks returns to sea level values.

While the increase in hemoglobin concentration augments arterial oxygen content, a number of changes detrimental to oxygen delivery may also occur. As the hematocrit approaches 60% triggered by the erythropoietic response, the viscosity increases while cardiac output and oxygen delivery decrease. This decrease in oxygen delivery is thought to be due to an impairment of perfusion of the microcirculation of the exercising muscles.²² In a study conducted by Schoene and Hackett on Mt. Everest in 1986, four climbers with hematocrits of 60% or greater underwent both psychometric and exercise testing before and after isovolumic hemodilution, which lowered hematocrits to 50%. Psychometric test performance improved while maximum exercise performance did not change. The implication from these results is that cerebral perfusion may have been improved, and that the decrease in arterial oxygen content did not diminish overall oxygen delivery.²³ The optimal hematocrit for high altitude acclimatization remains illusive, but clearly high-altitude survival and performance are impaired by an excessive polycythemic response.⁴

Schoene and Hornbein discuss the relationship that exists between oxygen and hemoglobin. They explain that when hemoglobin is more than 90% saturated with oxygen, there is an adequate partial pressure of oxygen (PaO_2) to maintain aerobic metabolism.

The transfer of oxygen from ambient air to tissue mitochondria is also influenced by the ease with which it is taken up by hemoglobin in pulmonary capillaries and released from hemoglobin in tissue capillaries. A number of variables affect the process, one of which is the affinity of hemoglobin for oxygen, as defined by the oxygen-hemoglobin dissociation equilibrium curve. An important feature of the oxygen-hemoglobin relationship is that over a large range of oxygen partial pressures (PaO_2 60 to 100 torr), hemoglobin is more than 90% saturated with oxygen. Thus, a change in PO_2 within that range has only a modest effect on arterial oxygen content. At PaO_2 less than 60 torr, hemoglobin saturation drops precipitously with small decreases in PaO_2 ; thus changes in PO_2 will result in relatively larger changes in blood oxygen content. At high altitude, certain adaptations of oxygen-hemoglobin affinity occur that may help facilitate oxygen transfer.⁴

Three substances in the blood influence the position of the oxygen-hemoglobin dissociation curve. These substances are H^+ , CO_2 , and 2,3-diphosphoglycerate (2,3-DPG). Increasing levels of these substances cause a right shift in the curve, allowing unloading of oxygen from hemoglobin to the tissues more easily. 2,3-DPG is generated within the red blood cell and is stimulated both by hypoxia and a hypocapnic alkalosis. Of the three substances mentioned above, the production of carbon dioxide and the subsequent generation of hydrogen ions at the tissues result in a greater unloading of oxygen; this is known as the Bohr effect.²⁴

At high to very high altitudes this rightward shift of the curve is counterbalanced by a respiratory alkalosis, which tends to shift the curve to the left, making it easier to load oxygen into the red blood cell in the pulmonary capillary of the lung. At extreme altitudes the effect of the severe respiratory alkalosis predominates, and a curve is shifted greater to the left. This left shift allows a significant increase in red blood cell hemoglobin-oxygen saturation for a given alveolar oxygen partial pressure. Ultimately this would create a higher arterial oxygen content. According to Schoene and Hornbein an ideal mechanism of adaptation was suggested by Reynafarge and colleagues in the alpacas and llamas of Peru, which possess hemoglobin with a high affinity for oxygen at the lung, but a low affinity at the tissues. This characteristic would appear to optimize both loading of oxygen in a hypobaric environment and unloading at the tissue level.⁴

ADAPTATION AT THE TISSUE AND CELLULAR LEVEL

Although not well researched in humans, a number of altitude-induced changes probably occur at the tissue and cellular level. The final transfer of oxygen from ambient air to the mitochondria involves the removal of oxygen from blood to the tissues. Several conduits within this portion of the oxygen cascade are dependent on the driving pressure of oxygen. The driving pressure of oxygen is essential for proper diffusion to occur from the capillary blood, across the vascular endothelium, and through the cytoplasm to the mitochondria where oxidative phosphorylation occurs. The process depends largely on diffusion and distance.

Certain adaptive changes take place to optimize oxygen diffusion from blood to the tissue during extreme hypoxia. These adaptive changes include: 1) minimizing the distance of diffusion of

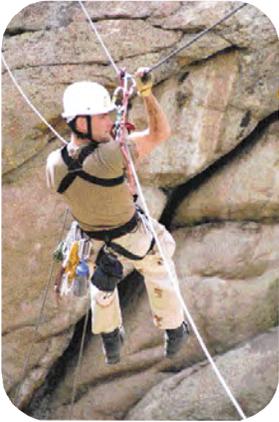
oxygen from the blood vessel to the mitochondria and 2) improving biochemical pathways in the mitochondria.⁴ A decrease in diffusion distance can be achieved by increasing the concentration of blood vessels in the tissues. Banchero, after conducting his studies in dogs with a three-week exposure to simulated altitude, documented an increase in capillary density and a decrease in the skeletal muscle size.²⁵

Some of the biochemical alterations that occur within the cell include elevated levels of myoglobin, which is an intracellular protein that binds with oxygen at very low tissue oxygen partial pressures. Myoglobin helps to facilitate the diffusion of oxygen from the blood to the mitochondria. Myoglobin concentration correlates quantitatively with the aerobic capacity of the muscle cell and is increased with training.²⁶ In 1977, Gimenz and colleagues discovered that myoglobin concentrations were elevated in animals native to high altitude and increased in dogs exposed to 435 torr for three weeks.²⁷ In addition to myoglobin, oxidative enzyme alterations during hypoxia have resulted in varied conclusions depending on the species, the tissue, and the stress. There appears to be an increase in the oxidative capacity of the enzymatic process (succinic dehydrogenase) enhancing long term adaptation to altitude. Schoene and Hornbein cite a number of studies that summarize these findings.⁴

Part two to follow in the Fall Edition!

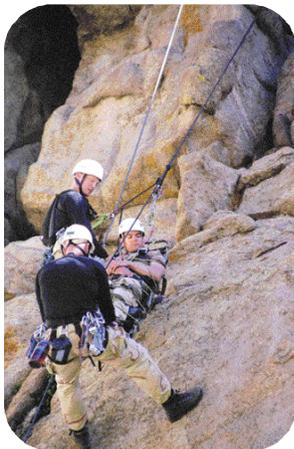
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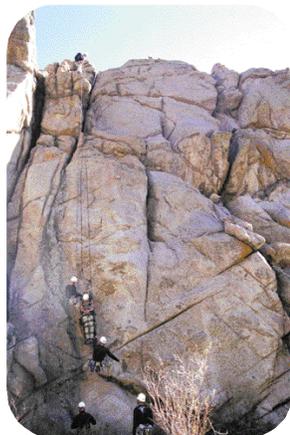
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Intubating Laryngeal Mask Airway versus Laryngoscopy and Endotracheal Intubation in the Special Operations Environment

Peter J. Cuenca, MD; Timothy S. Talbot, MD; David L. Nieman, PA-C; Paul M. Ryan, MD; Ian S. Wedmore, MD

ABSTRACT

This study plans to compare endotracheal intubation and the Intubating Laryngeal Mask Airway. First and foremost is airway management in trauma patients. Having a primary and alternative method of intubation, which is consistent and effective, is of utmost importance. This paper hopes to provide some insight into an airway management alternative through comparison in a very controlled environment.

Editors Note: The opinions expressed in this article are those of the authors and do not reflect the official policy of the Department of the Army, Department of Defense, or the U.S. Government.

INTRODUCTION

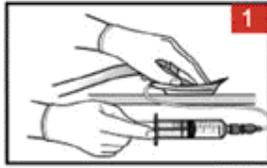
Airway management is the first and most important component in management of the trauma patient. This is especially true in the case of the combat casualty population. Ten percent of the injuries sustained in combat require immediate airway management prior to reaching a definitive care.¹ The gold standard for definitive airway control in the hands of experienced personnel is endotracheal intubation.²

Endotracheal intubation is a perishable skill and must be used on a regular basis to maintain proficiency. Advanced Trauma Life Support (ATLS) recommendations suggest that it not be taught or used by those who do not perform it on a regular basis. In-hospital endotracheal intubation by paramedics results in successful placement in only 46-87% of attempts although some EMS systems have reported out-of-hospital success rates of 90-95%.³⁻⁶ However, more recent data suggest that out-of-hospital intubations have a high misplacement rate. One Florida EMS system reported up to 25% misplaced endotracheal tubes, 67% of these were esophageal

intubations.^{7,8} In addition, airway management for trauma patients in tactical situations can make intubation by direct laryngoscopy difficult for even the most experienced providers. Under tactical conditions, with no ancillary support, every airway is a difficult intubation.

Recently, the Intubating Laryngeal Mask Airway (ILMA) has received attention as an effective tool for the management of the difficulty airway (DA). A review by Ferson et al. demonstrated a 96.5% success rate for blind intubation through the ILMA by physicians in patients with DA.⁹ Other studies with ILMA indicate a success rate of > 90% for blind intubation.¹⁰⁻¹⁶ A review of the literature revealed no study involving military personnel and utilization of the ILMA. A randomized controlled trial by Calkins and Robinson compared the ability of 11 Special Operations corpsmen and medics to learn to utilize the endotracheal tube, the laryngeal mask airway (LMA), and the esophageal-tracheal combitube (ETC) under combat conditions. Their results found insertion time for the LMA to be the shortest¹⁷, however; there was no examination of

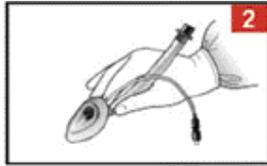
LMA-Classic™, LMA-Flexible™ & LMA-Unique™
Insertion Technique



Tightly deflate the cuff so it forms a smooth "spoon-shape." Lubricate the posterior surface of the mask with water-soluble lubricant.



Use the index finger to push cranially, maintaining pressure on the tube with the finger. Advance the mask until definite resistance is felt at the base of the hypopharynx.



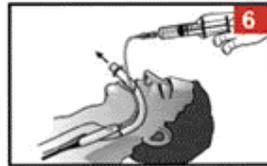
Hold the LMA like a pen, with the index finger placed at the junction of the cuff and the tube.



Gently maintain cranial pressure with the non-dominant hand while removing the index finger.



With the head extended and the neck flexed, carefully flatten the LMA tip against the hard palate.



Without holding the tube, inflate the cuff with just enough air to obtain a seal (to a pressure of approximately 60 cm H₂O). See in instruction manual for appropriate volumes. Never overinflate the cuff.

(Figure 2) Courtesy of LMA North America, Inc.

LMA-Fastrach™ Insertion Technique

To insert the LMA-Fastrach



Deflate the cuff of the mask and use a water soluble lubricant on the posterior surface. Rub the lubricant over the anterior hard palate.



Swing the mask into place in a circular movement maintaining contact against the palate and posterior pharynx. Never use the handle as a lever.



Inflate the mask, without holding the tube or handle, to a pressure of approximately 60cm H₂O

To insert the LMA-Fastrach Endotracheal Tube and Remove the LMA-Fastrach



Hold the LMA-Fastrach handle while gently inserting the lubricated ET tube into the metal shaft. The use of a standard, curved, PVC ET tube is not recommended.



Advance tube, inflate the ETT cuff and confirm intubation.



Remove the connector and ease the LNMA_Fastrach out by gently swinging the handle caudally. Use the stabilizing rod to keep the ETT in place while removing the LMA_Fastrach until the tube can be grasped at the level of the incisors.



Remove the stabilizing rod and gently unthread the inflation line and pilot balloon of the ETT. Replace the ETT connector.

ILMA use by military operators. Given the austere condition military combat casualty care providers operate under, as well as the high likelihood of difficult airways in trauma victims, it is important to determine the ability of military personnel to successfully intubate with the ILMA.

MATERIALS AND METHODS

We conducted a manikin study to determine the success rate of direct laryngoscopy/endotracheal intubation versus ILMA use by Special Operations medical personnel. Twelve Special Operations medics from the 2nd Battalion, 75th Ranger Regiment, all with Special Operations Combat Medics (SOCM) training (equivalent to EMT-P with enhanced ACLS, ATLS skills), participated in this one-day study. These medics had just completed annual refresher training in direct laryngoscopy three days prior to this study. A Laedral™ adult intubating manikin was used as the patient in this study. The Madigan Army Medical Center institutional review board approved this study.

During the first round of testing, each medic was placed in a lighted room and instructed to lie in the prone position in front of the intubating manikin. The prone position was chosen to simulate combat conditions. He was given one LMA-Fastrach™ ILMA Size 4 and one LMA-Fastrach™ endotracheal tube Size 7.5. The participants had not seen nor used the ILMA prior to the study. Each participant was given a one-page handout provided by LMA

North America that provided step-by-step instructions for placement of the LMA- Fastrach™ ILMA and insertion of the LMA-Fastrach™ endotracheal tube (ILMA ETT) (Figure 2).

They were given no other information about the study, manikin or airway device. Each participant had ten minutes to read the handout. No feedback or questions were permitted prior to or during the initial round of testing. The posterior portions of the ILMA and the ILMA ETT were prepared as per the instruction hand out with SURGILUBETM Water soluble lubricant. One 20cc syringe was attached to the ILMA cuff and a 10cc syringe was attached to the ETT cuff. A standard bag valve mask was also provided. Participants had two separate attempts to intubate the manikin using the ILMA. All attempts were timed using a digital timer. Our main outcomes were success or failure of each intubation attempt and time to successful intubation/ventilation. Success of intubation/ventilation was defined as placement of the endotracheal tube in the trachea, inflation of the ETT cuff, and successful ventilation of manikin lungs. This was assessed via direct visualization of the tube in the trachea through the bottom of the manikin, as well as palpation and visualization of the manikin's inflated lungs by one of the investigators. If time exceeded 60 seconds, the attempt was recorded as a failure. If the ILMA ETT was placed in the esophagus, the medic was instructed to stop and the attempt recorded as a failure. These criteria were used for the remainder of the study.

Participants were given a FOREGGERTM Folding Scope, a size 4 Miller blade from Lite-Blade™, and a size 7.5 Portex™ endotracheal tube. This same equipment was used throughout all of this study's iterations. Medics were given two attempts each to intubate the manikin using direct laryngoscopy. All attempts were timed using digital timer. Intubation was conducted from the prone position lying in front of the manikin. Our main outcomes were the same as those for the protocol described above.

Following completion of the first iteration of testing, all of the participants received a 10-minute demonstration of ILMA insertion and endotracheal tube placement. A two minute review of airway anatomy using a teaching manikin model was also conducted. The soldiers' question regarding the ILMA and ILMA intubation were answered. The medics were retested using the same size 4 LMA-Fastrach ILMA and size 7.5 ILMA ETT with the protocol previously described. All attempts occurred

from the prone position in a well-lit room. Times were again recorded as above. Our main outcomes were the same as those for the protocol above.

The participants were asked to perform intubation using the ILMA in dark conditions. Each medic was taken into a completely dark room. Prior to the light being turned off, the medic was placed in the prone position in front of the intubating manikin. The soldier was allowed to set the ILMA and ILMA ETT with syringes attached to the side of the manikin. A small penlight was placed at the side of the manikin's head with the light turned on. The lights were then shut completely off, with the penlight serving as the only light source for the soldier. Each participant was given two attempts to place the ILMA ETT. Outcomes as described previously were recorded.

After completing this iteration, the medics performed two attempts at intubation using direct laryngoscopy in a completely dark room. Again, each soldier was placed in the prone position in front of the intubating manikin. The laryngoscope and 7.5 Portex™ endotracheal tube with syringe in place were set next to the manikin. There was no penlight for this iteration. The lights were then turned off and the room was completely dark. The participant was then given two attempts at placement of the ETT. Outcomes as described previously were then recorded.

DATA ANALYSIS

Data was then entered into an Excel Spreadsheet and the mean and median intubation times for each iteration were calculated. The data was analyzed using STAT VIEW for WINDOWS 4.5 using a student paired t-test to calculate p-Values for the data, mean times to intubation, range and standard deviation.

RESULTS

Twelve people participated in the study. All were paramedic certified. All had conducted airway training using direct laryngoscopy three days prior to the study. Five of the twelve participants had more than six live human intubations using direct laryngoscopy within the past year. Three of the twelve had no human intubations in the past year. All of the paramedics tested had at least one live animal intubation within the last year. None of the soldiers tested in this study had ever seen an ILMA used prior to the study. Of the twelve soldiers who participated in the study, only ten completed it. Two medics were

unable to complete the night portion of the study to other operational responsibilities. However, these two soldiers were able to conduct the first two iterations of the study, ILMA intubation untrained and direct laryngoscopy.

On the first iteration of the study (untrained ILMA intubation in the prone position in lighted conditions), there was an initial success rate of 79.2% (19/24 attempts were successful). Of the 5 attempts that were intubation failures, two were secondary to esophageal intubation and three were secondary to exceeding the 60-second time limit (62.3, 69.8, and 65.7 seconds). The mean time to ILMA intubation, including attempts exceeding sixty seconds, was 31.9 seconds. The mean time to ILMA intubation, excluding failures, was 26.5 seconds. The median time for ILMA intubations, including attempts exceeding sixty seconds, was 26.5 seconds. The median time for ILMA intubations, excluding failures, was 26.2 seconds. The second iteration of the study (direct laryngoscopy in the prone position in lighted conditions) demonstrated a success rate of 91.7% (22/24 attempts successful). Of the two attempts that were intubation failures, both exceeded

ing attempts exceeding sixty seconds, was 15.1 seconds. The median time for ETT intubations, excluding failures, was 14.8 seconds.

On the third iteration of the study (trained ILMA intubation in the prone position in lighted conditions), there was a success rate of 100% (20/20 attempts were successful). The mean time to ILMA intubation was 22.9 seconds and the median time was 21.5 seconds. The fourth iteration of the study (trained ILMA in the prone position in dark conditions) demonstrated a success rate of 100% (20/20 attempts successful), a mean time to intubation of 24.5 seconds, and a median time of 22.5 seconds.

Finally, the fifth iteration of the study (direct laryngoscopy in the prone position in dark conditions) demonstrated a success rate of 100% (20/20 attempts successful), a mean time to ETT intubation of 25.1 seconds, and a median time of 23.5 seconds. (For a summary of study results refer to Table 1.) Direct laryngoscopy was significantly faster in daylight conditions with both untrained ($p = .0055$) and trained ($p = 0.5$) with a mean difference of 12.9 seconds and 2.4 seconds respectively. ILMA intubation was faster at night but did not reach statistical

	ILMA Untrained Daylight	Direct Laryngoscopy Daylight	ILMA Trained Daylight	Direct Laryngoscopy Night	ILMA Trained Night
Success rate (Intubation in 60 sec.)	79.2% (19/24)	91.7% (22/24)	100% (20/20)	100% (20/20)	100% (20/20)
Mean Time to intubation (sec) Including times > 60 sec	31.9	20.6	22.9	25.1	24.4
Standard Deviation (sec)	14.7	14.1	9.1	10.0	6.4
Range (sec)	19.6 to 69.8	19.6 to 60.5	12.1 to 50.4	12.7 to 47.4	12.8 to 36.5

the time limit (60.5 and 60.3 seconds). The mean time to ETT intubation, including attempts exceeding sixty seconds, was 20.6 seconds. The mean time to ETT intubation, excluding failures, was 17.0 seconds. The median time for ETT intubations, includ-

ing attempts exceeding sixty seconds, was 15.1 seconds. The median time for ETT intubations, including failures, was 14.8 seconds.

Table 1

Success rate, mean time to intubation (all times) using direct laryngoscopy and the ILMA by 12 Special Operations Paramedics in daylight and

night condition using a manikin model. Also included are the associated standard deviations and ranges.

DISCUSSION

British anesthesiologist Archie Brain developed the LMA. After careful study of the plaster

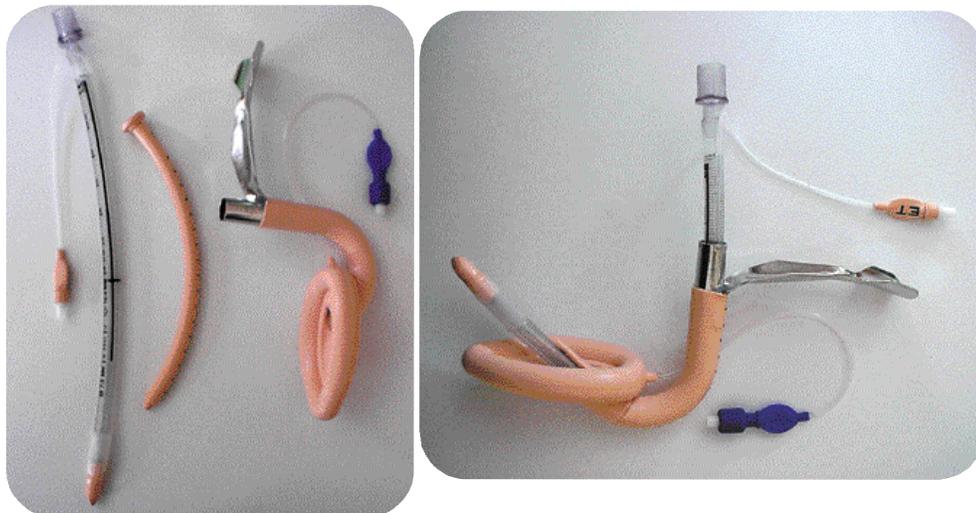


Figure 1: A. Intubating LMA Size 4 with 7.5 mm ETT and stylet. B. Insertion of ETT with elevation of the Epiglottis Elevation Bar.

casts of cadaver airways, Brain found that inflation of an elliptical cuff in the hypopharynx created an airtight seal around the posterior larynx. By adapting a Goldman dental mask with 10mm tubing, he created the first LMA prototype, which was successfully used on a patient in 1981.^{18,19} The first clinical trial of the LMA occurred in 1983.²⁰ The LMA became commercially available in the United States in 1992 and is currently used by pre-hospital personnel in the United Kingdom, Japan, and increasingly in the United States.^{21,22} The LMA provides a rapid, secure airway in many patients who cannot be intubated, and is recognized by the American Society of Anesthesiologists Task Force of Management of the Difficult Airway as an important option in non-surgical DA management.²³ The ILMA was introduced as a modification of the classic LMA in 1997. In addition to serving as an emergent ventilating device, the ILMA was designed to allow blind intubation.²⁴ Since its introduction to use, there have been numerous case reports and studies documenting effective ventilation and intubation in DA cases using the ILMA.

The ILMA design allows for blind tracheal intubation (*Figure 1*):

1. A short anatomically curved, rigid steel shaft that follows the oral, pharyngeal, and laryngeal axes.
2. A metal handle providing control of the ILMA in the patient's airway as well as the ability to manipulate and maintain position for blind placement of the endotracheal tube.
3. A V-shaped guiding ramp at the end of the ILMA that directs the endotracheal tube (ETT) towards the glottis.
4. An epiglottis elevating bar which lifts the epiglottis up while the ETT is pushed through the trachea.²⁵

These features allow the head and neck of a patient to remain in the neutral position during intubation. Walt et al. demonstrated that the ILMA caused less extension of the neck at C1-2 and C2-3 (area of greatest cervical excursion during intubation) than intubation by direct laryngoscopy.²⁶ In trauma patients in whom cervical spine movement is limited or undesirable, the ILMA is a safe, fast, effective method of securing the airway.

Numerous studies show that unskilled personnel can insert a standard LMA more rapidly and reliably than an endotracheal tube, and that they can ventilate more effectively with a LMA compared to bag and mask.²⁷⁻³¹ A study using ILMA and a manikin model found that on first attempted use of the ILMA and with no prior training, physicians had a 83% success rate. With training, there was a 100% success rate.³²

The special operations environment ranges from isolated small unit actions up to battalion sized direct action missions across a range of environmental conditions not seen in conventional unit warfare. Intubation of casualties on the battlefield is a controversial subject. Some would advocate that there is

no role for intubation in a multiple casualty scenario and, with this, the authors would agree. However, there are specific mission profiles in which intubation may be indicated. Development of a special operations soldier is expensive and actions to maintain life even in the severely wounded are indicated when possible and given adequate operational conditions. In situations with single to few casualties, extended exfiltration times, and adequate personnel support, intubation of a casualty may be indicated.

Given the current propensity to exploit our superior night fighting prowess, medical intervention on a casualty is done in low or no light conditions on the ground, in the back of a vehicle, or aircraft. Airway maintenance must be accomplished without the medic becoming a casualty as well. It also requires the ability to control the airway from a variety of unconventional positions not seen in the standard ambulance or operating room. Even in optimal conditions with sufficient personnel, intubation success rates among paramedics range from 46-94% with unrecognized improper placement in 25% in the prehospital environment in civilian EMS systems.³⁻⁸ Correct endotracheal intubation in difficult airway situations has an even lower success rate in spite of optimal environments. Given the limitations of the operational environment, all airways must be considered difficult airways, and we must optimize the ability of the medic to correctly place the endotracheal tube to ensure survival of the indispensable special operations soldier.

There is evidence in the literature supporting use of the ILMA for management of the difficult airway. However, there is no study to date evaluating use of the ILMA in military trained combat casualty medical personnel. Given these facts, this study provides new and important information regarding ILMA and its future in airway management on the battlefield. The ILMA allows for easy learning with little training, rapid placement, and minimal deterioration of skills over time. These attributes greatly enhance the combat care provider's ability to manage the airway of casualties in the austere environment of combat operations.

Our study suggests that the ILMA is inherently easy to learn how to use. With only limited training (reading a one page handout), Special Operations medics had a 79.2% success rate intubating from the prone position in a lighted environment compared to the 91.7% success rate with direct laryngoscopy. Their mean time to intubation was approx-

imately 10 seconds slower than intubation using direct laryngoscopy under the same conditions (both including failure times and excluding failure times). After only 12 minutes of instruction, average time for intubation with the ILMA improved by 9 seconds and the success rate for intubation was 100%. Under dark conditions, the ILMA had a 100% success rate of intubation as did direct laryngoscopy. Intubation with the ILMA was equivalent to direct laryngoscopy with no significant difference in placement times. This is even more significant given that none of the participants had ever seen an ILMA prior to the study day, while the participants had had approximately two days of refresher training on direct laryngoscopy three days prior to the study. Ease of use and simplicity are important for airway devices. As mentioned previously, direct laryngoscopy is perishable skill. It relies upon extensive training in evaluating and recognizing airway anatomy. Lack of experience, may make intubation with direct laryngoscopy difficult to impossible, especially under the difficult conditions found on today's battlefield. The simplicity and ease of use of the ILMA as demonstrated by this study warrant its use as an airway adjunct by military personnel, especially medical care providers in the Special Operations arena.

This study has several limitations to this study in regard to equipment and training methods. One limitation of this study is that it is a manikin study. Difficulties found in combat casualties, such as redundant neck tissue, down folded epiglottis, concomitant face/head/neck trauma, and vomiting and aspiration, were not present in the manikin model. Use of the manikin model prevents us from evaluating these potential problems and their effect on ILMA intubation. However, these are factors, which affect the ability to adequately visualize the airway anatomy, and would have a profound effect on the ability to intubate using standard endotracheal intubation. The ILMA is a blind intubating technique, which is not dependent on anatomy recognition and visualization, therefore these factors may not impact the ability to successfully intubate and ventilate. There is literature regarding only LMA use that suggests manikin-only training is equivalent to LMA training using both manikins and live patients.^{33, 34} Not requiring participants to select the proper size ILMA was another limitation of this study. Proper size selection is key to ILMA intubation with use of a size 4 for less than 70 kg and size 5 for greater than 70 kg.³⁵ Improper size selection

may lead to intubation failure. The ability to intubate within 60 seconds was an artificial limit set by the investigators to enhance the rigorous environment. In other arena's, intubation would not take place until approximately 90-120 seconds. This decrease in time resulted in the majority of the failures noted and may create a higher false negative rate.

Another limitation is the number of subjects available for evaluation. Due to the current operations, and the shortage of qualified medics available, the authors attempted to maximize numbers available by tailoring the study protocol to current training schedules. However, given our positive results, a much larger trial should be done in the future to validate these results. We suspect the ILMA will demonstrate even better results in Special Operations medics with minimal direct laryngoscopy training. This could be done in conjunction with the Joint Special Operations Medical Training Center upon initial entry of soldiers into the special operations medical training program.

In conclusion, our data suggests that the ILMA, in the hands of the Special Operations Combat Medic, is a simple to use device requiring minimal airway training, and it could serve well as a primary airway control device in the special operations setting.

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UNCONVENTIONAL WARFARE



UNCONVENTIONAL MEDICINE

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There I was	Correspondence	Editorials
SOMA Update	Photo Gallery	Med Quiz
Dedication		

What is your least favorite section of the JSOM? (Circle one)

Departments	Component Surgeon Offices	Education & Training
Features	Research & Development	Legacy
There I was	Correspondence	Editorials
SOMA Update	Photo Gallery	Med Quiz
Dedication		

What improvements would you make to the JSOM?

FOLD ALONG THIS LINE

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CONTINUING MEDICAL EDUCATION TEST

NO.1

Heat-Associated Illness

JSOM



1. In an effort to dissipate heat, the body distributes blood flow between muscle, skin and viscera depending on exertion, endogenous heat production and environmental temperatures.
T or F
2. When exposed to a combination of heat and regular exercise the body starts to adapt with the following physiological changes.
 - A. Increased cardiac stroke volume
 - B. Decreased sweat rate
 - C. Lower sweat sodium content
 - D. a & c only
 - E. b only
 - F. all the above
3. While training in hot weather it has been found that the body's metabolic rate during a road march will jump to 6 times the basal rate and as much as 12 to 14 times for those who are running.
T or F
4. Factors that can influence the occurrence and severity of heat illness include:
 - A. Temperature, humidity, solar and wind conditions
 - B. Physical fitness and body type
 - C. Heat retention from darkly shaded clothing
 - D. a & c
 - E. a & b
 - F. all the above
5. Eating large meals or missing meals can predispose you to heat illness or may effect acclimatization.
T or F
6. In regards to water and electrolyte replacement:
 - A. Build up for tolerance against dehydration should be conducted slowly
 - B. High sugar solutions may impede water absorption
 - C. Water and salt replacement should be conducted hourly
 - D. Loss of about 1 liter of water has already occurred by the time a person senses they are thirsty
 - E. a & c
 - F. b & d
7. Rectal temperatures are used by many health care facilities to differentiate heat illness syndromes, but is unreliable alone for assessing the severity of heat illness.
T or F

8. Exertional heat stroke signs and symptoms include:
 - A. Orthostatic manifestations
 - B. Confusion, delirium, obtundation and/or coma
 - C. Red, hot and dry skin
 - D. a & c
 - E. a & b
 - F. all the above

9. The absence of delirium, obtundation, or coma can in no way distinguish exertional heat injury from exertional heat stroke.
T or F

10. Prompt treatment of heat stroke includes rapid body cooling and rehydration.
T or F

Answer Sheet on page 54

CONTINUING MEDICAL EDUCATION TEST

NO.2

Going Beyond Thin Air: Part 1

A Comprehensive Review for The Special Forces Dive Medical Technician

JSOM



- 1) The reduction in partial pressure of oxygen becomes physiologically significant due to the increased barometric pressure in ambient air as you ascend in altitude.
T or F
- 2) Although altitude exposure can be classified into 5 types and differentiated by altitude itself, arterial hemoglobin saturation in a persons body will remain relatively unchanged despite movement through out these ranges.
T or F
- 3) Acclimatization, a physiological adaptation to altitude includes the following:
 - A. cardiovascular, respiratory, renal and blood vessel response
 - B. increased erythropoiesis and oxygen delivery to tissues
 - C. both a & b
 - D. only a
- 4) A greater yield of cellular ATP produced via the glycolic pathway enables the body to maintain an aerobic state under hypoxic stress at higher altitudes as the body acclimatizes.
T or F
- 5) The general rate at which a person can acclimatize depends on:
 - A. rate of ascent and altitude attained
 - B. whether or not an individual had previously accelerated the acclimatization process
 - C. individual physiology of a person
 - D. both a & c
 - E. c only
 - F. all the above
- 6) The first consequence of the reduction in alveolar oxygen caused by the reduction of partial pressure of oxygen at high altitudes stimulates peripheral chemoreceptors adjacent to these structures:
 - A. aortic arch
 - B. carotid body at the bifurcation of common carotid artery
 - C. neither a or b
 - D. only a
 - E. both a & b

- 7) Oxygen flux is dependent upon several variables, these include:
- A. pressure gradient for oxygen from the alveolus to the pulmonary capillary blood
 - B. surface area for the gas exchange
 - C transport of calcium at the smooth muscle membrane
 - D. only a
 - E. a & b
 - F.. all the above
- 8) As a compensatory action during the acclimatization process, loss of plasma volume is accompanied by a relative increase in hemoglobin concentration and an improved oxygen carrying capacity of the blood.
T or F
- 9) The basic hematological adaptations that occur during acclimatization are as follows:
- A altered affinity of hemoglobin for the oxygen molecule
 - B erythropoetin stimulation
 - C polycythemia
 - D a & b
 - E. a & c
 - F. all the above
- 10) An adaptive change the body undergoes to optimize oxygen diffusion from the blood to the tissue, is increasing concentration of blood vessels in the tissue itself.
T or F

Answer Sheet on page 55

Continuing Education Evaluation Form
Journal of Special Operations Medicine, Volume 2, Edition 2

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POST-TEST – Answer Sheet

Article 1 Heat-Associated Illness Page 15

Please circle the letter that corresponds to the correct answer:

- | | |
|----------------|----------------|
| 1. T F | 6. a b c d e f |
| 2. a b c d e f | 7. T F |
| 3. T F | 8. a b c d e f |
| 4. a b c d e f | 9. T F |
| 5. T F | 10. T F |

Article 2 Going Beyond Thin Air Page 32

Please circle the letter that corresponds to the correct answer:

- | | |
|----------------|----------------|
| 1. T F | 6. a b c d e |
| 2. T F | 7. a b c d e f |
| 3. a b c d | 8. T F |
| 4. T F | 9. a b c d e f |
| 5. a b c d e f | 10. T F |

**Continuing Education Evaluation Form
Journal of Special Operations Medicine
Volume 2 Edition 2 Spring 02
Date of Original Release 31 May 02**

	Article 1 Page No.15					Article 2 Page No. 32				
	Strongly Agree		Strongly Disagree			Strongly Agree		Strongly Disagree		
	5	4	3	2	1	5	4	3	2	1
Educational Value:										
I learned something new that is important.	-	-	-	-	-	-	-	-	-	-
I verified some important information.	-	-	-	-	-	-	-	-	-	-
I plan to discuss this information with colleagues.	-	-	-	-	-	-	-	-	-	-
Readability Feedback:										
I understood what the authors were trying to say.	-	-	-	-	-	-	-	-	-	-
Overall, the presentation of the article enhanced.	-	-	-	-	-	-	-	-	-	-
My ability to read and understand it.	-	-	-	-	-	-	-	-	-	-

Were the educational objectives of the article(s) met? YES ___ NO ___ YES ___ NO ___

If no, please explain: _____

Do you think that the article(s) unduly emphasized one company's products? YES ___ NO ___ YES ___ NO ___

Comments: _____

How long did it take to complete Article 1? ___ minutes **Article 2?** ___ minutes

What changes will you make in your practice as a result of reading the article(s)?

I hereby certify that I have read the article(s) of the activity identified above and am eligible to claim credit. Print Name: _____
Signature: _____
Date: _____



Case Report: Initial Psychotic Break Associated with Exertional Heat Stroke: Report on Three Cases and Discussion

Robert Ambach, PA (SEAL)

Jessie Gross, PA (SEAL)

Larry Garsha, MD

INTRODUCTION

Heat stroke is well documented to be an elevated core temperature associated with neurologic findings. Psychosis has many reportable causes to include neurologic, endocrine, metabolic, fluid or electrolyte imbalances, hepatic or renal diseases, autoimmune, and psychiatric disorders. A review of the literature for hyperthermia/heat stroke cross-referenced with psychosis displayed only those cases associated with the administration of neuroleptics and anesthetics. We were unable to find any references in regards to exertional heat stroke and an acute psychotic break. Three such cases presented at the Naval Special Warfare Center in a six-month period. The patients were students enrolled in the Basic Underwater Demolition/SEAL Training at Coronado, California. We feel they are important to document.

CASE REPORTS

Case 1

A 19-year old white male was brought to the medical clinic after suffering a loss of consciousness during a run on the beach. His initial core temperature per rectum was 104.1°F. Patient arrived in the clinic disoriented. He was cooled to eutermic temperature and transferred to the local emergency department. The patient began exhibiting combative behavior shortly after arriving at the ED and was sedated and intubated. Patient was evaluated with Computerized Axial Tomography, lumbar puncture, and blood chemistries. All studies were normal. Patient was admitted to the ICU for observation. After extubation patient attempted to physically attack three different nurses. The psychiatry depart-

ment evaluated patient. Their differential diagnosis was an idiopathic schizophrenia type psychotic disorder or a psychotic disorder due to a psychotic break from the physical and psychological stress of the heat stroke and treatment.

Case 2

A 21-year old white male brought to the medical clinic under the control of six servicemen. Patient was very combative, speaking in inappropriate statements. Patient had just completed military physical training. He was found in the shower acting strange. His core temperature per rectum was 102.9°F. Patient was cooled to eutermic temperature. He was given Droperadol, as he was still very combative after being cooled. Patient was taken to the local emergency department where he was evaluated with Computerized Axial Tomography, lumbar puncture, and blood chemistries. The only remarkable lab was a CK of 1026. Patient awoke with a normal mental status and demeanor after the Droperadol wore off. Patient was admitted for observation and released the following day. Patient remembers little about events prior to and in the clinic. Psychiatric evaluation led to a diagnosis of a psychotic break secondary to hyperthermia.

Case 3

A 19-year old white male suffered a loss of consciousness during a run and was brought to the medical clinic. Initial core temperature per rectum was 107.0°F. Patient was combative and required physical restraints while being cooled. Patient was

cooled to a eutermic temperature at which time his demeanor was normal as well as his mental status. Patient was transported to the local emergency department where he was further evaluated. Patient was admitted for observation and treatment of rhabdomyolysis. This patient had a CK of 2924. The patient's hospital stay was uneventful, and he was released the following day. Five months later the patient was involved in a motor vehicle accident, and he admitted to smoking marijuana and stated he wanted to die. He was evaluated by the psychiatry department and admitted to the hospital for treatment of accident injuries as well as an acute psychotic episode. Further psychiatric evaluation found the patient to have suffered a severe manic episode with psychotic features. The patient was placed on lithium to prevent future episodes. Diagnosis at the time of his second admission was Psychotic Disorder NOS. His discharge diagnosis was Bipolar Disorder, Type I, most recent episode Manic, Severe With Psychotic Features. Patient was discharged on Eskalith with directions to follow-up as an outpatient. He was subsequently separated from the military.

Psychological testing consisting of the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) and the Personality Assessment Inventory was conducted on the patients in cases 1 and 2. Both patients fell within the normal ranges in all categories. The patient in case 3 was not tested as he suffered his heat stroke before the testing was conducted and was disenrolled from the program.

DISCUSSION

Numerous case reports have been documented on people experiencing a psychotic break during a hyperthermic event. These cases are primarily patients who suffered from malignant hyperthermia from anesthesia or who developed malignant neuroleptic syndrome from taking anti-psychotic medication. A literature review failed to locate any documentation of people suffering from an acute psychotic break from exertional heat stroke.

Body temperature is a balance between heat production and heat dissipation. Heat is produced from metabolic processes and heat absorption. Heat is dissipated from conduction, convection, radiation, and evaporation. Thermal regulation is controlled by the anterior hypothalamus. The autonomic nervous system causes vasodilation for convection, radiation, conduction, and sweating for evaporation when the core temperature rises. Conversely, the

hypothalamus causes vasoconstriction in the skin and increases muscle tone or shivering when the core temperature drops.¹ All of these responses are involuntary.

The aforementioned processes maintain the hypothalamic set-point. Hyperthermia results when heat production exceeds heat dissipation. Fever results when circulating cytokines increase the set-point.² There are numerous types of hyperthermia from as many different causes, and there appears to be a subtle link between some of them.

Exertional heat stroke (EHS) is a result of extreme muscle exertion. It presents with an increase in core temperature, altered mental status (delirium, stupor, or coma), hypotension, tachycardia, hyperventilation, and rhabdomyolysis.³ It is typically treated by administration of intravenous fluids, evaporative cooling measures, and cold water baths. The mortality rate is about 10%.

Malignant hyperthermia (MH) is caused by an increase in the release of calcium from the sarcoplasmic reticulum, resulting in severe muscle hypermetabolism. The most common triggering agents are halogenated inhalation agents and depolarizing muscle relaxants. There is typically a family history of MH due to an autosomal dominant genetic mutation. MH usually presents with hyperthermia, severe muscle rigidity, hypotension, tachycardia, hyperventilation, and rhabdomyolysis. The treatment for MH is the use of dantrolene (a muscle relaxant), and physical cooling. The mortality rate is high. The incidence of MH is approximately 1 in 50,000 people this is based on genetic statistics.⁴

Neuroleptic malignant syndrome (NMS) is typically preceded by the use of a neuroleptic agent (i.e. phenothiazines, butyrophenones, and thioxanthenes; haloperidol is the most common). It occurs in about 0.2% of patients taking neuroleptics.⁵ NMS may be triggered by a blockade of dopaminergic receptors at the corpus striatum. The usual presentation is a patient with hyperthermia, muscle rigidity, altered mental status, hypotension, tachycardia, and rhabdomyolysis. The treatment for NMS is to discontinue the triggering agent. The use of dantrolene has been shown to increase survival. Physical cooling may also help. The mortality rate is approximately 20%.

There have been numerous cases that report elevations in temperature associated with psychological stress. Anticipatory anxiety has been documented as being one of the causes for an elevation in

temperature.⁶ Studies have found that this type of fever is not affected by the use of cyclo-oxygenase inhibitors but rather is associated with benzodiazapine use.

Other causes of hyperthermia include dehydration, which can cause volume depletion, which results in a decrease in cutaneous vasodilation and a decrease in the patient's ability to sweat.¹ Salicylate intoxication, anticholinergic and psychotropic medications can contribute to hyperthermia. Illicit drugs such as cocaine and amphetamines have been shown to place the patient at significant risk for developing heat stroke through increased heat production.⁷ Alcohol decreases heat tolerance. Creatine monohydrate increases functional energy production, resulting in increased metabolic heat production.⁸ Hormonal imbalances can precipitate hyperthermia. Thyrotoxicosis is the most common hormonal cause of hyperthermia and is often precipitated by stress. Pheochromocytoma, adrenal insufficiency, hypoglycemia, and hyperparathyroidism can also cause hyperthermia.⁵

It is possible that patients with EHS and MH may have a similar underlying skeletal muscle abnormality that involves a dysregulation of the myoplasmic calcium cycling. This being the case, EHS could possibly respond to dantrolene.⁹ It has been suggested that physical exercise and emotional stress might also be a trigger for MH and possibly EHS.¹⁰

Some people have shown a predisposition to more extreme sympathetic nervous system activation and/or dysfunction in response to emotional or psychological stress. This may constitute vulnerability for NMS.¹¹ The hallmark of NMS is an altered mental status. The altered mental status of NMS can be expressed as mania, a sense of doom, overwhelming anxiety, and possibly psychosis.

While psychogenic fever has been shown to be a cause of hyperthermia, it appears that most cases were limited to fevers of less than 104°F. Therefore, this may be more of a contributing factor than an actual cause in the development in several types of hyperthermic events.¹²

It is possible the physiological changes associated with hyperthermia affects people the same way regardless of the cause of the hyperthermia. Therefore, hyperthermia could be a risk factor to the cause of some psychosis and conversely some psychological conditions could predispose some people to hyperthermic conditions.¹³

There have been other cases of psychosis in

BUD/S trainees who never had a reported case of heat stroke. Those patients could have developed their psychosis from other causes, or they could have suffered a subclinical case of heat stroke during or prior to BUD/S training and had a delayed onset of their psychosis.

CONCLUSION

The etiology of psychosis is arguable in the three patients in this paper. It is indisputable, however, that they all developed a significant psychosis after suffering a significant heat injury during one of the U.S. military's most stressful courses. This chronological association and the absence of documented cases of people developing psychosis after suffering an exertional heat stroke places greater importance on these interesting and tragic cases.

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FINAL TRIBUTE TO BUDDY RICHMOND

Steve Yevich and Len Blessing

A friend is measured by the quality of time, not the quantity. A friend provides memories for a lifetime, especially when those memories are intense and focused like they can get when in Special Forces. While friends maintain the memory, and smile within about those times in their lives, and maybe yearn for the 'good old days'- they know those days are forever gone.

I had a friend, Buddy Richmond. You may not know my Buddy Richmond, but you probably had a "Buddy Richmond" when you were in SF who came along during the single most influential and formative period of your life. We were young, idealistic, lived high on emotion, played hard, fought hard, believed like children in our Country, and wondered about God. We all were torn between being like the chivalrous knights from fairy tales and Hollywood, versus being the unscrupulous fighters who our Camp MacKall mentors trained us to be -- doing ANYTHING necessary to survive and win - the rogue-warriors who were Special Forces back then, and on which the Green Beret legend was built. There is an endless stream of memories, but foremost in my mind are the extreme ones that tied us together in an unspoken allegiance and dedication to each other. We were *The X-Treme Games*, decades before the notion was ever discovered by TV-land - and MY Buddy always was working on the edge, daring us to follow.

It all began during our Special Forces training at Ft Bragg in 1969. In the Special Forces training group, we sweated and worked harder than we ever had before, taught by those grizzled "old" multi-tour Viet Nam vets (Tom Jones, Duke Snyder, and many other now-nameless characters), ever-attentive because we knew we were bound for combat in Viet Nam. On after-duty time, Buddy was always an instigator, jolting us out of our dulled senses to face "situations" which he would just create in the usual settings in Fayette-Nam -- places like Caruso's Lounge, the Circus, the Pink Panther, and a few dozen other bars which I only knew by the shapes of

their neon signs, never knew they had actual names. With Viet Nam looming on everyone's horizon and making us desperate to capture Life, this was where we left our first marks, blood, cash, youthful idiocy and testosterone. When SF training was over, we "ghosted" at Holding Company for several long months, going crazy on rumors and the never-ending waiting, until we received orders diverting us to 10th Special Forces Group at Ft. Devens, MA. Within a few months, Buddy and I were the only two lucky ones of our class whose persistent calls and begging to "Mrs. A" resulted in orders to Viet Nam. Making the trip across the continent on our way to the Ft. Lewis overseas processing point, we took control of a scene in a college bar in Rhode Island, defending my Uncle Paul against a couple of equally drunk "kollige kids". We were invincible, we had nothing to lose - we were Green Berets - and we were going to die in Viet Nam. As we drove across the US, we dropped down into Tijuana and ended up running for our lives one morning around 0200h, realizing that some people were more invincible than us.

Those high charged times rapidly became very sobering when we hit Nha Trang, where we both unhesitatingly volunteered for CCN, going to CCN Launch sites as new Chase Medics. Our mentors were the Woodhams and John Gross - and we rapidly became members of that small group which flew every insertion and every extraction, peering tensely over the belly edge, watching green and red tracers, and colored smoke, and Willie Pete fireworks from exploding rockets, waiting for the call to jump off and enter, disoriented, into the chaotic dimension on the ground. How did we run so fast, but stay so low to the ground that our shirt buttons dragged? ...firing back into bush, rarely seeing targets, only gunsmoke. Remember those sexy, but useless Uzi's we carried for a while, until we relegated them to PX visits only? Remember the time Pappy Budrow let me leave Quang Tri to see you at the Phu Bai Launch site -- and a team called for a "routine" extraction, so we each chose a different chopper and

flew out, and the VC hit the team while you were on the chopper pulling them out? We each flew in under fire to pull out hits - but mine was dead - we laughed about that, and argued whether he was dead before or after I took care of him. Oh, there was another time Woodham got hit while going in on a desperate RT, and I spent that night on the ground with a Bright Light team looking for Dehnke and Hollinshead's (?name?) team, and we were ambushed that morning and took 3 hits. Boy, I don't think I ever told you how good it was to see you on the chopper that flew in under fire to pick up my wounded. That was the best sight ever! Time almost stopped.

My Buddy Richmond, like yours, was much more than a medic; he was the true SF warrior. He was heroic, and unrewarded - but he let it go. There was the time Buddy was in the chopper that landed under fire to pick up four Montanyards from an RT, and the chopper crashed from 30 feet up -- the pilot shot through the face, and the co-pilot wounded badly. Although thrown, Buddy had so much experience on the ground doing team extractions that he immediately and naturally took over without a second thought. Downed in dense undergrowth in triple canopied jungle, Buddy organized the two door gunners and the Yards, pulled out the wounded co-pilot, and using that tiny survival radio contacted Covey and led off the chopper nose to a postage-stamp LZ for pick up. Truly an unrecorded feat in

bravery and survival.

Our lives may have gone separate ways but we were glued together through the SF bond, and bolted and welded through the CCN Chase Club membership. Now, since 29 MAY 02, there's one less guy in our tiny club who knows what it really meant to fly Chase - who knows what it took for a Woodham to climb down a 30 foot ladder to a wounded guy while flying at 100 knots at 8000 feet - what it's like to spend hours on the details of packing both "pows" and meds so both have top priority - what the adrenaline rush feels like when you hear "...RT in trouble, launch!" -- what extremes of emotions those Hughey turbines bring when they start winding up on a hurried start - and what it feels like to transit from freezing in the open chopper listening on the headset to a frantic RT on the ground, to jumping out with a 90 pound rucksack from a ten foot hover to face flying brass and tight faces, and the smell of cordite, smoke, jungle-stench, and sudden quiet. Damn, Buddy, you just took a chunk of my heart when you died.

Buddy, your picture hangs with us, your Special Ops medic buddies, in the Joint Special Operations Medical Training Facility at Ft. Bragg NC. This is where our journey began - and you're now part of The Legend in the gallery. We'll all get together here as a team, and we'll stay here, together as a team, in final rest.





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WOUND IRRIGATION

The primary determinants of infection are bacterial counts and amount of devitalized tissue remaining in the wound. Ridding a wound of bacteria and other particulate matter requires more than soaking and gentle washing with a disinfectant. Irrigating the wound with a forceful stream is the most effective method of reducing bacterial counts and removing debris and contaminants. The cleansing capacity of the stream depends on the hydraulic pressure under which the fluid is delivered. Irrigation is best accomplished by attaching an 18 or 19-gauge catheter to a 35 cc syringe or a 22-gauge needle to a 12 cc syringe. This creates hydraulic pressure in the range of 7 to 8 lb/in² and 13 lb/in², respectively. The solution is directed into the wound from a distance of 1 to 2 inches at an angle perpendicular to the wound surface and as close to the wound as possible. The amount of irrigation fluid will vary with the size and contamination of the wound, but should average no less than 250 ml. Remember: "The solution to pollution is dilution."

There is a lack of consensus on which irrigation solution is the best for open wounds. Those who subscribe to the dogma that nothing should enter a wound that could not be instilled safely into the eye believe that normal saline is the best solution. In a study of 531 patients with traumatic wounds, there was no significant variation in infection rates among sutured wounds irrigated with normal saline, 1 % povidone-iodine, or pluronic F-68 (Shur-Cleans).

Tap water was recently found to be as effective for irrigating wounds as sterile saline. In fact, the infection rate was significantly lower after irrigation with tap water, and no infections resulted from the bacteria cultured from the tap water.

Improvised wound irrigation requires only a puncturable container to hold the water, such as a sandwich or garbage bag, and a safety pin or 18-gauge needle (see Box 18-1).

WOUND CLOSURE

Before a wound is closed, all foreign material and grossly devitalized tissue should be

BOX 18-1 RECOMMENDED TECHNIQUE FOR WOUND IRRIGATION

1. Fill a sandwich or garbage bag with water.
2. Disinfect the water with iodine tablets, iodine solution, or povidone-iodine or by boiling it.
3. Normal saline can be made by adding 2 teaspoons of salt (9 g) per liter of water.
4. Seal the bag.
5. Puncture the bottom of the bag with an 18-gauge needle, safety pin, fork prong, or knife tip.
6. Squeeze the top of the bag forcefully while holding it just above the wound, directing the stream into the wound.
7. Use caution to ensure that none of the irrigation fluid splashes into your eyes.

removed. Debridement can be done with scissors, a knife, or any other sharp object.

Wounds can be closed with sutures, staples, tape, pins, or glue. Although suturing is still the most widely used technique, stapling and gluing are ideal methods for closing wounds in the wilderness.

Clinical studies of the use of staples to close traumatic lacerations have found various advantages of stapling over suturing: wound tensile strength is greater, there is less inflammation, the time required for closure is shorter, and fewer instruments are needed. Most important, the cosmetic outcome is not compromised. Staplers are light-weight, pre-sterilized, and easy to use.

WOUND TAPING

Skin tapes are useful for shallow, nongaping wounds and have several advantages over suturing, including reduced need for anesthesia, ease of application, decreased incidence of wound infection, and availability. Any strong tape can be used to improvise skin tape strips, but duct tape works especially well (see Box 18-2). Puncturing holes in the tape before application helps to prevent exudate from building up under the tape.

Although benzoin is usually applied to the skin before the tape to augment adhesion, cyanoacrylate glue (Crazy Glue) works better. If benzoin is

BOX 18-2
WOUND TAPING TECHNIQUE

1. Obtain hemostasis and dry the wound edges.
2. Apply benzoin or cyanoacrylate glue to the skin adjacent to the wound. Benzoin should be left to dry until it becomes tacky, but the tape should be applied to the glue while the glue is still wet.
3. Tape should be cut to quarter-inch or half-inch widths, depending on the size of the laceration, and to a length that allows for 2 to 3 cm of overlap on each side of the wound.
4. Secure one half of the tape to one side of the wound. Oppose the opposite wound edge with a finger while the tape is secured to the other side.
5. Wound tapes should have gaps of 2 to 3 mm between them to allow for serous drainage.
6. Cross-stays of tape can be placed perpendicular over the tape ends to prevent them from peeling off.
7. Additional glue can be applied to the tape edges every 24 hours to reinforce adhesion.

available, the two can be used in combination.

Wound taping does not work well over joints or on hairy skin surfaces unless the hair is first removed. Scalp lacerations can sometimes be closed by tying opposing strands of hair to approximate the wound edges.

GLUING

Although Histoacryl (butyl-2-cyanoacrylate) tissue adhesive is frequently used in Europe and Canada for sutureless skin closure, it has not yet been approved by the Food and Drug Administration for use in the United States. When applied to the skin surface, Histoacryl provides strong tissue support and peels off in 4 to 5 days without leaving evidence of its presence. It provides a faster and less painful method for closing lacerations than does suturing and has yielded similar cosmetic results in children with facial lacerations. Histoacryl evokes a mild acute inflammatory reaction with no tissue necrosis.

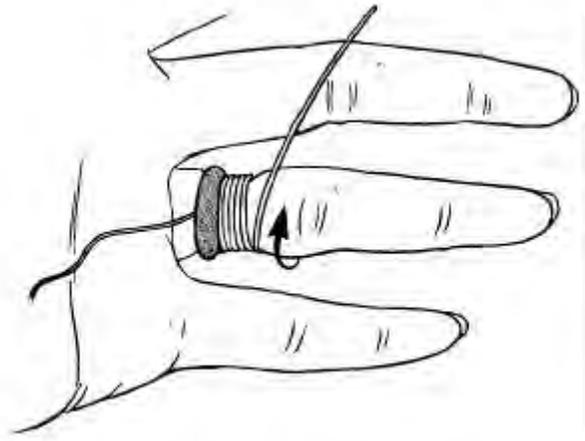
Histoacryl has also been used successfully to treat superficial painful fissures of the fingertips ("polar hands"), which commonly occur in cold climates and at high elevations.

Crazy Glue or Super Glue (ethyl-2-cyanoacrylate) is a shorter chain cyanoacrylate derivative that is readily available in the United States. Compared with Histoacryl, it releases more heat during polymerization and produces a severe acute

inflammatory reaction, with tissue necrosis and a chronic foreign body giant cell response. Its use for routine wound closure cannot be recommended at this time.

RING REMOVAL

Rings should be removed quickly from injured fingers and after any trauma to the hands. Progressive swelling may cause rings to act as tourniquets. If a ring cannot be removed with soap or lubricating jelly, the string wrap technique can be employed. A 20-inch length of fine string, dental



floss, umbilical tape, or thick suture is passed between the ring and the finger. The string is pulled so that most of it is on the distal side of the digit and then is wrapped around the swollen finger from proximal to distal. The wrapping should begin next to the ring and continue past the proximal interphalangeal joint. Successive loops of the wrap should be placed close enough together to prevent any swollen skin from bulging between the strands. The ring is removed by unwinding the proximal end of the string and forcing the ring over the distal string. If the string is not long enough, the technique may require repeated wraps.

IMPROVISATIONAL TOOLKIT

Some people, convinced they could whittle a Swan- Ganz catheter from a tree branch if they had to, enter the wilderness with nothing more than a Swiss Army knife. However, a little foresight and preparation make improvisation much easier. Efficiency translates into speedy preparation and assembly, which ultimately results in better patient care. The following section lists items that will make improvisation easier in the field.

KNIFE

The knife can be a fairly simple model, but it should have an awl for drilling holes into skis, poles, sticks, and so on. The awl on a Swiss Army knife works quite well for this purpose. This allows the rescuer to create well-fitted components during improvisation (for example, a drilled cross-bar attached to drilled ski tips for an improvised rescue toboggan.)

TAPE

Some form of strong, sticky, waterproof tape should be carried. (This is one item that cannot be improvised.) Either cloth adhesive tape (already in the medical kit) or duct tape should be used. Duct tape is ideal for almost all tasks. It can even be used on skin when needed (for example, to close wounds, treat blisters, or tape an ankle). Some persons may be sensitive to the adhesive. Fiberglass strapping tape has greater tensile strength and is ideal for joining rigid components, such as taping two ice axes together. However, it is less sticky than duct tape and not as useful for patching torn items. Extra tape can be carried by wrapping lengths of it around pieces of gear.

PLASTIC CABLE TIES

Lightweight cable ties can be used to bind almost anything together instantaneously (for example, binding pack frames together for improvised litters or ski poles together for improvised carriers). They are also perfect for repairing many items in the back country.

PARACHUTE CORD

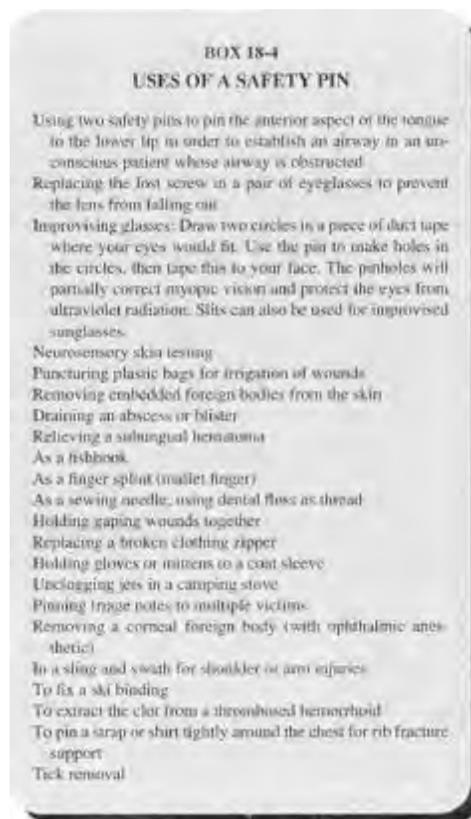
Parachute cord has hundreds of uses in the back country. It can be used for trucker's hitch traction and for tying complex splints together. Parachute cord is light, and a good supply should be carried.

SAFETY PINS

Safety pins have a variety of uses (see Box 18-4).

WIRE

Braided picture-hanging wire works well because it is supple and ties like line. Its strength makes it superior for repairing and improvising components under an extreme load, such as fabricating improvised rescue sleds or repairing broken or detached ski bindings.



BOLTS AND WING NUTS

Bolts and wing nuts make the job of constructing an improvised rescue sled much easier (see section on rescue sleds). Bolts will be useful only if holes can be created to put them through. Therefore a knife with an awl is needed for drilling holes through skis, poles, and so on.

PREFABRICATED CROSS-BAR

The prefab cross-bar can be used for double ski pole traction splint systems. A cross-bar can be easily fabricated in the field from a branch or short section of the patient's ski pole, but carrying a prefabricated device such as a 6-inch predrilled ski pole section saves time (Fig. 18-10).

ENSOLITE (CLOSED-CELL FOAM) PADS

Since the introduction of Therm-a-Rest-type inflatable pads, closed-cell foam has become increasingly scarce; however, closed-cell foam (Ensolite) is still the ultimate padding for almost any improvised splint or rescue device. The uses for closed-cell foam are virtually unlimited. Even die-hard Therm-a-Rest fans should carry a small amount of closed-cell foam, which doubles as a comfortable seat cushion and is lightweight. Furthermore, unlike

inflatable pads, Ensolite will not puncture and deflate.

Therm-a-Rest pads also have their place. They can be used as padding for many long bone splints and immobilizers (one example is an improvised universal knee immobilizer). An inflatable pad can also be used to cushion pelvic fractures. First the deflated pad should be wrapped around the pelvis. Then the pad is secured with tape and inflated, creating an improvised substitute for military antishock trousers (MAST device).

FLUORESCENT SURVEYOR'S TAPE

Surveyor's tape can be used much like Hansel and Gretel's bread crumbs to help relocate a route into or out of a rescue scene. It is also ideal for marking shelters in deep snow and can serve as a wind sock during helicopter operations on improvised landing zones. Surveyor's tape is not biodegradable, so it should always be removed from the site after the rescue is completed.

SPACE BLANKET OR LIGHTWEIGHT TARP

For improvising hasty shelters in times of emergency, some form of tarp is essential. In the snow a slit trench shelter can be built in a matter of minutes using a tarp. Otherwise, the complex and time-consuming construction of improvised structures such as snow caves, igloos, or tree branch shelters might be necessary. Typically, little time or help is available for this task during emergencies. In addition, tarps are essential for "hypothermia wraps" when managing injured patients in cold or wet conditions. The only advantage of a space blanket over other tarps is its small size, which means there is a good chance it was packed for the trip.

SAM SPLINT

Introduced in 1985, the SAM splint (Fig. 18-22) has largely filled the niche formerly occupied by military-style ladder splints and wire mesh splints. The SAM splint is exceptionally versatile. It weighs approximately 41 fl ounces and rolls up into a space approximately the size of a Kerlix bandage. It is made of a thin sheet of malleable aluminum sandwiched between two thin layers of closed cell foam. The splint initially has no rigidity, but after structural U-shaped bends are placed along the axis of the splint, it becomes quite rigid.

IMPROVISED EYEGLASSES

Exposure of unprotected eyes to ultraviolet radiation at high altitudes may result in photokeratitis (snow blindness). Symptoms are delayed, and the victim is often unaware that an eye injury is developing. When sunglasses are lost at 14,000 feet in the snow, photokeratitis can develop in a mere 20 minutes. Improvised sunglasses can be fashioned from duct tape, cardboard, or other light-impermeable material that can be cut. Cardboard glasses with narrow eye slits can be taped over the eyes for protection.

Slits can also be cut into a piece of duct tape that has been folded over on itself with the sticky sides opposing. After a triangular wedge is removed for the nose, another piece of tape can be applied to secure the glasses to the head.

Pinhole tape glasses can improve vision in a myopic person whose corrective lenses have been lost. With myopia, parallel light rays from distant objects focus in front of the retina. The pinhole directs entering light to the center of the cornea, where refraction (bending of the light) is unnecessary. Light remains in focus regardless of the refractive error of the eye (Fig.18-23). Pinhole glasses decrease both illumination and the field of vision. Therefore a piece of duct tape or cardboard should be punctured repeatedly with a safety pin, needle, fork, or other sharp object until enough light can enter to focus on distant objects, and the device should be secured to the face.



Fig. 18-22 -The SAM splint consists of a thin sheet of malleable aluminum sandwiched between two thin layers of foam. It can be easily molded to injured extremities and becomes rigid after U- shaped bends are placed into the aluminum.



The JSOM is one of the best read journals housed here. Thanks again.-----MH-----

Margaret J. Harrison, Librarian
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We are an Area Support Medical Company with a Treatment Platoon and Ambulance Platoon. By MTOE we have Physicians, Physician Assistants, a Nurse and 91B/91W enlisted personnel. In view of our mission, I believe your publication would be an invaluable tool for teaching our soldiers and sustaining their level of training. Please consider our request.
SFC Howard H. Kittell II

I am currently an Army HPSP medical student at Tulane Medical School and a former active duty Combat Engineer Officer. I have been borrowing copies of the Journal of Special Operations Medicine since its initial publication. I was wondering if it is possible for me to get a subscription or back issues.
LT Guy Hill

Announcements

I have completed all of my research and my outline for my book titled I'm the one called "Doc" . My book, however, is not another book about vietnam. It is not a novel. It is a book of poetry; poems such as mine, written by various authors, their story of what inspired them to write the poem, combat artist sketches depicting the poems, information on the artists, and factual information about the U.S. Navy Hospital Corps, and a lot more. If you know of anyone who has written a poem about Navy Hospital Corpsmen, doctors, nurses, etc., I would like to get in touch with them. I speak from over 30 years of experience and sharing this with others is what I want to do.

Thank you for your dedicated efforts.

Harry "Doc" Penny, HMC(AC)USN(Ret)

This is a reminder that the PJs are having a reunion Sept 12-14, 2002, in Albuquerque, New Mexico,
<http://www.pjsinnam.com/PJ%20Reunion.htm>.

I'm sure there are going to be a least a couple hundred of PJ combat medics there with a lot of stories. This a source that the Journal of Special Operations Medicine would be interested in for submission into "There I Was"!

Wayne Fisk

Apologies/Corrections

In Vol. 2 Ed 2 Spring 02, pg 1--the last paragraph of Col Hammer's remarks were cut off and the first paragraph was inadvertently reprinted. It was supposed to read: Finally, I want to briefly state that we are close to resolving other issues for our SOF Medics;-Hemoglobin Oxygen Carrying Fluid is getting lots of attention and we hope to soon have this in place of crystalloid fluids and Hetastarch as volume expanders.

-Factor VII research goes on.

-A one-handed tourniquet is on the way.

-Interosseus fluid routes in place of fragile Intravenous routes are being examined for our use.

So, again, find your colleagues and get information to us that will help you in the medical care and salvage of our SOF Warriors-they are truly National Treasures.

In Vol.2, Ed 1 Winter 02, pg.58--the date on the picture of CSM Dickinson in the hammock should be 1964 instead of 1965.

Medical Direction and SOF

Warner Anderson MD

Medical direction, also called medical control, is a cornerstone of emergency medical services. Medical direction is the foundation of EMS in the sense that it allows emergency medical technicians and other EMS personnel to provide services to the sick and injured. In essence, the medical director is responsible for the content and quality of care provided by EMS.

QUALIFICATIONS

A medical director is, by definition, a physician who is a licensed medical doctor or osteopathic physician (M.D. or D.O.) in the jurisdiction (e.g. state or territory) from which the EMS operates. Most jurisdictions require the medical director to attend a formal course in medical direction within a certain time frame after accepting the post. This time period is usually a matter of months.

The medical direction course is generally three or four days long. It describes the responsibility of medical direction and the statutory requirements entailed. The medical directors are instructed in the scopes of practice of the different EMS providers, such as first responder, EMS dispatcher, and EMT-basic, intermediate, and paramedic. For medical directors of flight EMS units, direction may also include registered nurses and nurse specialists (such as neonatal intensive care), physician assistants and physicians.

Interestingly, there is no firm requirement that a medical director have hands-on experience or ride-along time. Most medical directors probably come from a background involving some EMS, but a number have little direct EMS patient care experience. Thus, specialized field gear such as Combitube®, LMA®, traction splints, hard or vacuum splints, backboards, extrication devices, AEDs, transport ventilators and so on may be foreign to the medical director.

Clearly, the conscientious medical director will inform himself of the indications and cautions

of these devices, but this is a self-motivated matter. When performing quality of care studies, a medical director who is not familiar with the tools of the trade can be of little use.

Similarly, there is generally no statutory requirement for a medical director to actually observe care provided in the field. A medical director may choose to do ride-alongs or have other means, such as a dedicated vehicle, to observe training, but again, this is an issue of self-motivation. Obviously, a medical director learns much about his EMS agency while observing care. Only rarely should the medical director do hands-on direct care, however, since the EMTs are a team and outside interference can cause confusion in care sequencing.

ON-LINE VERSUS OFF-LINE

On-line medical control is that provided by the receiving hospital ED or other facility during patient treatment and transport. It is usually over radio, but may be over cell phone or satellite phone. In remote areas, it may even be by closed circuit television. This control should be by physicians who are knowledgeable in the protocols and capabilities of the EMTs. More often, it is by ED nurses. However, as a matter of policy, all critical patient on-line control should be by physician.

Contrast off-line medical control, which is the province of the medical director. EMTs must be able to operate independently of on-line medical control in case of dire emergency, communications failure, or disaster. In this case, they must have authorized protocols for each likely emergency problem.

PROTOCOLS

Treatment protocol development is a major responsibility for any medical director. Since care is provided under the legal guidance of the medical director, protocols reflect his sense of appropriateness for the area served. For example, what works in Tampa may not work at all deep on the Navajo

Reservation. Interventions such as endotracheal intubation may be less important when the transport time is ten minutes, but critical when the transport time is two hours.

Many local protocols are available, and most states have regional or state-wide protocols available for consideration. These may be applicable as published, or may require extensive revision. For example, a published protocol may allow an intermediate EMT to administer morphine for myocardial infarction, congestive heart failure or pulmonary edema. The intermediate also is allowed naloxone (Narcan®) as a narcotic reversal agent. However, the published protocol does not allow the intermediate to give morphine for other types of pain. A medical director in a rural area, then, may seek a special skill waiver to allow the intermediate to administer morphine in otherwise-uncomplicated long bone fracture with long transport times.

While a special skill waiver may seem like a minor thing, it quickly spirals into a big thing. For a sizeable EMS to initiate the seemingly-simple intervention of giving morphine in a fracture, the medical director and whatever training personnel are involved must insure that the morphine is not given in head injury, and probably not in abdominal injury. Should it be given to a patient in shock?

Define shock clinically in a manner that fits the EMS environment: tachycardia? Low systolic or mean arterial pressure? Who does not have tachycardia once their femur has been broken? Who can reliably take a systolic blood pressure in a bouncing ambulance with a writhing patient?

The point demanding answers on the special skill application and implementation, then, grows in complexity, far beyond simply deciding "Pain? Morphine good."

Similar questions arise when special skills are requested for intermediates to do endotracheal intubations, or for paramedics to do rapid sequence intubation. For the intermediate doing intubations, the medical director must consider whether the EMT has been trained on an am manikin, and whether this is sufficient. If not, will animal surrogates serve adequately? Cadavers? Or are live humans necessary to demonstrate competency? And then, how many intubations must be performed annually to maintain competency? In the end, will intubation help or harm more patients?

The new medical director rarely appreciates the complexity of the ambush awaiting him just down the trail.

EQUIPMENT

EMS is a trendy field, about as trendy as middle California. Just as everyone needs a goat cheese pizza, so everyone needs the latest biomedical device. The medical director will be approached on all sides by EMTs and salespeople touting the latest (and most expensive) devices.

"Here's a spring-loaded gun that shoots a big-bore I.V. needle into the patient's sternum, but it doesn't actually stab them in the heart."

How do you know it's safe in the hands of your worst EMT? If a medical director cannot be certain, then the product should be avoided.

"Here's a tube you can blindly insert into the apneic patient's throat and then it's foolproof that you can ventilate through it."

Then the medical director must react to the first death due to esophageal tear that comes across his desk from "Risk Management".

Beyond these issues of appropriateness, the medical director must deal with establishing appropriate change when such change may be resisted because of budgetary or institutional inertia. For example, if a vacuum backboard is more comfortable than a rigid one, but costs \$400 more and has a life expectancy of six months, the medical director must decide how hard to push for its implementation. Now, what if the EMTs don't like it because it takes twenty-five seconds more to properly put the patient into it, and the agency's administration will crucify them if they don't recover it from the ED immediately upon patient transfer?

The clinical indications may often be clear to the medical director, while the financial or operational indications are just as clearly different to management or the EMTs.

In the end, the medical director must have a good knowledge of the literature (scientific, not sales), of practices in nearby communities, and some sense of how to prioritize needs against resources and operational constraints.

QUALITY OF CARE

The major problem confronting the medical director in quality of care issues is simply stated: Half of all EMTs are going to be below average.

However, just because an EMT is "below average" doesn't mean he's incompetent. In fact, an underlying tenet of quality improvement holds that incompetent workers should be few and far between in a healthy system. When a problem arises, first

turn your attention to the system.

Is the system conducive to good performance? The EMTs must have adequate protocols, equipment, a good work environment, reasonable morale, sufficient training and continuing education, and just enough challenge to keep them wanting to improve and expand their skills.

EMT performance must have checks and balances - the partner EMT, on-line medical control, other agencies' EMTs, police, firefighters, and emergency department personnel. When an EMT is dangerously incompetent, these observers will alert the medical director. He must then decide whether the actions in question were isolated and accidental, a correctable educational or skill deficiency, or a malicious or willful act.

When an incident is result of a malicious act, or a dangerous incompetence, then the medical director must have the power to remove the EMT from patient care until adjudication is made or educational fixes are implemented.

When an incident is the result of "unprofessional behavior", the agency's management or the licensing department takes the lead as the disciplinary body. Here, the medical director must work in close cooperation with these administrative authorities in an advisory capacity.

Just as important as individual performance reviews, generic studies highlight potential problem areas and try to abort problems before they become serious. Certain types of runs should always be reviewed by the supervisory EMT and the medical director: patient refusal of service or complaint, death, codes, threats of litigation, unusual runs,

departures from protocols, interference by another agency or health care personnel, and others as prescribed by state standards.

The focused study is the most fruitful - the supervisory EMT and the medical director choose a type of run that is of special interest and review all those runs for a period. For example, they may review all pediatric codes, snakebite or all head injuries. This type of review is best done with any other involved agencies, such as dispatch, the ED, or other involved parties. Then the agencies develop a plan to correct any problems emerging from the studies.

Finally, in the case of each of the quality of care interventions above, the medical director must be able to answer "How did the intervention improve, or not improve, care."

In the end, in any EMS the medical director is the person responsible for the content and appropriateness of the care at the scene and in transport. The medical director vets the EMTs' qualifications and performance, and addresses problems as they are identified. The medical director draws upon the resources of the agency's administration, the licensing authority, educational institutions, and sister agencies and hospitals to improve the entire system.

In many regards, the medical director is the resident expert. He has limited powers but immense moral authority, which he must exercise in the interest of his patients and his service.

He doesn't drive the train or blow the whistle, but if it jumps the tracks, see who gets the attention.

Photo Gallery



SOF Medics treating POW in Afghanistan
Photos courtesy of USASOC Sine Pari



Navy Hospital Corpsman 3 David Island, Special Operations Combat Medics course student, charges out of a scum-brown moat with a "wounded" classmate during a base-defense exercise at Camp Mackall, Fort Bragg, NC
Photos courtesy of USASOC Sine Pari

JSOMTC NY Rotation//Aug 99



Dr giving instructions to students while in training in NY C
Photos courtesy of USASOC Sine Pari

Student obtaining blood gases
Photos courtesy of USASOC Sine Pari



Student Starting an IV
Photos courtesy of USASOC Sine Pari



Dedication



Chief Petty Officer Matthew J. Bourgeois, 35, of Tallahassee, Fla., was killed while in direct support of Operation Enduring Freedom on 28 March 2002. Chief Petty Officer Bourgeois, a SEAL Hospital Corpsman, stepped on a land mine killing him and wounding another service member while conducting small unit training in a remote location near Qandahar, Afghanistan.

Stationed in Norfolk, Va., Chief Bourgeois was deployed to Afghanistan in support of Operation Enduring Freedom and received his fatal wounds after apparently stepping on an enemy ground emplaced munitions, which appears to have been a land mine. The accident, came at a time of increased combat activity as the winter snows melted in the rugged Afghan mountains. This highlights the dangers troops face even when not under direct hostile fire.

The blast occurred at a demolition range next to the compound that once housed former Taliban leader Mullah Mohammed Omar, according to local government spokesman Yusuf Pashtun. Several U.S. special forces troops live in the compound.

An Afghan guard said U.S. troops had been collecting confiscated weapons and ammunition and storing them at the compound for disposal. He said he heard a series of six explosions about noon that Monday.

Gen. Richard Myers, chairman of the Joint Chiefs of Staff, said the accident shows "our servicemen and women remain at risk."

U.S. military spokesman Maj. Bryan Hilferty at Bagram air base north of Kabul, said about 10 soldiers were disposing of the rockets when the accident happened. He said the injured soldier was flown to the U.S. military base just south of Kandahar, where American authorities said his injuries were not life-threatening.



Photo provided to the U.S. Navy by Bourgeois family.

Special Forces Aidman's Pledge

As a Special Forces Aidman of the United States Army, I pledge my honor and my conscience to the service of my country and the art of medicine. I recognize the responsibility which may be placed upon me for the health, and even lives, of others. I continue in the caring for the sick and injured. I know "Primum non nocere" (First, thou shalt do no harm) and I will follow the competent medical authority. These confidences which come to me in secret. I recognize my responsibility to medicine such knowledge of its art and practice to improve my capability to this purpose. As an American soldier, I have determined ultimately to place above all considerations of self the mission of my team and the cause of my nation.



I profess the limitation of my skill and knowledge and promise to follow the maximum and to seek the assistance of more whenever it is available. My attendance on the sick, I will treat as impart to others who seek the service of medicine as I possess, and I resolve to continue to

Pararescue Creed

I was that which others did not want to be. I went where others feared to go, and did what others failed to do. I asked nothing from those who gave nothing, And reluctantly accepted the thought of eternal lonlinessshould I fail. I have seen the face of terror; felt the stinging cold of fear, and enjoyed the sweet taste of a moment's love. I have cried, all, I have lived times others would be able to say, that I was proud of what I was: a P.J.



And I resolve to continue to place above all considerations of self the mission of my team and the cause of my nation.

~ author unknown~

It is my duty as a Pararescueman to save a life and to aid the injured.

I will perform my assigned duties quickly and efficiently, placing these duties before personal desires and comforts.

Navy Poem

I'm the one called "Doc"... I shall not walk in your footsteps, but I will walk by your side. I shall not walk in your image, I've earned my own title of pride. We've answered the call together, on sea and foreign land. When the cry for help was given, I've been there right at hand. Whether I am on the ocean or in the jungle, Giving it Sailors or Marines. aid to my fellow man, be corpsman and you think So the next time you see a think of the job he's doing of calling him "squid", And if you ever have to as those before him did. go out there and your life is on the block, Look at the one right next to you... I'm the one called "Doc".



~ Harry D. Penny, Jr.

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